ETIOLOGICAL AND CLINICAL PROFILE

OF

GLOMERULAR DISEASES IN ADULTS

Dissertation submitted to

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

in partial fulfillment for the Degree of

DOCTOR OF MEDICINE - BRANCH I

GENERAL MEDICINE

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TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVEL – 11 , TAMILNADU

CERTIFICATE

This is to certify that the Dissertation entitled **"ETILOGICAL AND CLINICAL PROFILE OF GLOMERULAR DISEASES IN ADULTS**" submitted by **Dr.VINOJ. A** to The Tamilnadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment for the award of M.D.Degree (GENERAL MEDICINE) is a bonafide work carried out by him under my guidance and supervision during the course of study 2015-2018. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other.

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DECLARATION

I solemnly declare that the dissertation titled **"ETIOLOGICAL AND CLINICAL PROFILE OF GLOMERULAR DISEASES IN ADULTS"** is prepared by me. The dissertation is submitted to the Tamilnadu Dr.M.G.R.Medical university towards the partial fulfillment of requirements for the award of M.D. Degree (Branch I) in General Medicine . I also solemnly declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any university, found either in India or abroad.

Place: Tirunelveli.
Date:

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I sincerely thank all the **PATIENTS** who cooperated with me for participating in the study.

Last but not the least, I thank **ALMIGHTY GOD**, for giving me wisdom, favours and blessings.

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CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO:895/GM/2016

PROTOCOL TITLE : ETIOLOGICAL AND CLINICAL PROFILE OF GLOMERULAR DISEASES IN ADULTS.

PRINCIPAL INVESTIGATOR: Dr. A.VINOJ, MBBS

DESIGNATION OF PRINCIPAL INVESTIGATOR: POST GRADUATE IN GENERAL MEDICINE

DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear , Dr. A. Vinoj, The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 05.08.2016.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

Dr.K.Shanmuraman MD
Registrar, TIREC
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CERTIFICATE - II

This is certify that this dissertation work title **"CLINICAL AND ETIOLOGICAL PROFILE OF GLOMERULAR DIESEAS IN ADULTS"** of the candidate **Dr.A.VINOJ** with registration Number **201511359** for the award of M.D. in the branch of **GENERAL MEDICINE.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **12 PERCENTAGE** of plagiarism in the dissertation.

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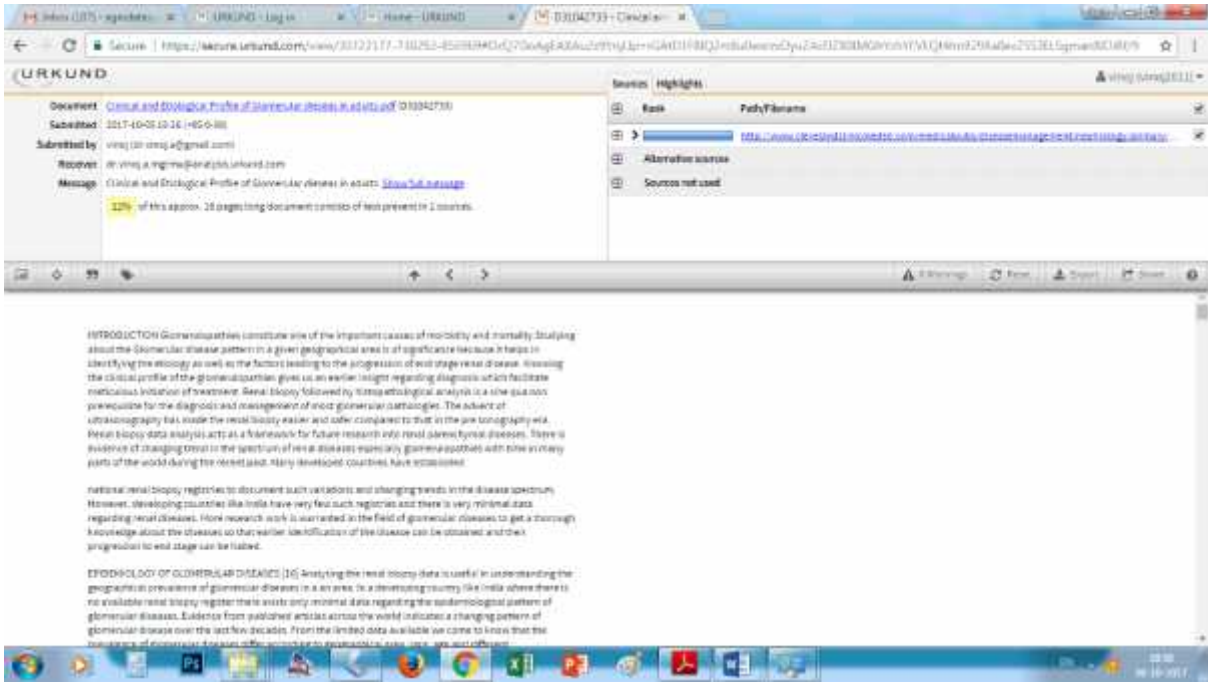
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INTRODUCTION Glomerulopathies constitute one of the important causes of morbidity and mortality. Studying about the Glomerular disease pattern in a given geographical area is of significance because it helps in identifying the etiology as well as the factors leading to the progression of end stage renal disease. Knowing the clinical profile of a glomerulopathies gives us an earlier insight regarding diagnosis which facilitate meticulous initiation of treatment. Renal biopsy followed by histopathological analysis is a sine qua non prerequisite for the diagnosis and management of most glomerular pathologies. The advent of ultrasonography has made the renal biopsy easier and safer compared to that in the pre sonography era. Renal biopsy data analysis acts as a framework for future research into renal parenchymal diseases. There is evidence of changing trend in the spectrum of renal diseases especially glomerulopathies with time in many parts of the world during the recent past. Many developed countries have established
national renal biopsy registries to document such variations and changing trends in the disease spectrum. However, developing countries like India have very few such registries and there is very minimal data regarding renal diseases. More research work is warranted in the field of glomerular diseases to get a thorough knowledge about the diseases so that earlier identification of the disease can be obtained and their progression to end stage can be halted.
EPIDEMIOLOGY OF GLOMERULAR DISEASES [16] Analyzing the renal biopsy data is useful in understanding the geographical prevalence of glomerular diseases in a an area. In a developing country like India where there is no available renal biopsy register there exists only minimal data regarding the epidemiological pattern of glomerular diseases. Evidence from published articles across the world indicates a changing pattern of glomerular disease over the last few decades. From the limited data available we come to know that the

CONTENTS

INTRODUCTION

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Renal biopsy followed by histopathological analysis is a sine qua non prerequisite for the diagnosis and management of most glomerular pathologies. The advent of ultrasonography has made the renal biopsy easier and safer compared to that in the pre sonography era. Renal biopsy data analysis acts as a framework for future research into renal parenchymal diseases.

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More research work is warranted in the field of glomerular diseases to get a thorough knowledge about the diseases so that earlier identification of the disease can be obtained and their progression to end stage can be halted.

EPIDEMIOLOGY OF GLOMERULAR DISEASES [10]

Analysing the renal biopsy data is useful in understanding the geographical prevalence of glomerular diseases in a an area. In a developing country like India where there is no available renal biopsy register there exists only minimal data regarding the epidemiological pattern of glomerular diseases.

Evidence from published articles across the world indicates a changing pattern of glomerular disease over the last few decades.

From the limited data available we come to know that the prevalence of glomerular diseases differ according to geographical area, race, age and different histopathological pattern existing in different regions of the world.

Primary glomerulonephritis (PGN) has the maximum prevalence in India. Among the PGN cases Among the PGN cases, the most prevalent one is the minimal change disease followed by focal segmental glomerulosclerosis.

Secondary glomerular disease (SGN) is the second prevalent among which lupus nephritis followed by amyloidosis and diabetic nephropathy are the most prevalent diseases.

Other conditions like Tubulointerstitial disease and vascular diseases are less common. The incidence of FSGS and IgAN has been increasing since 1999.

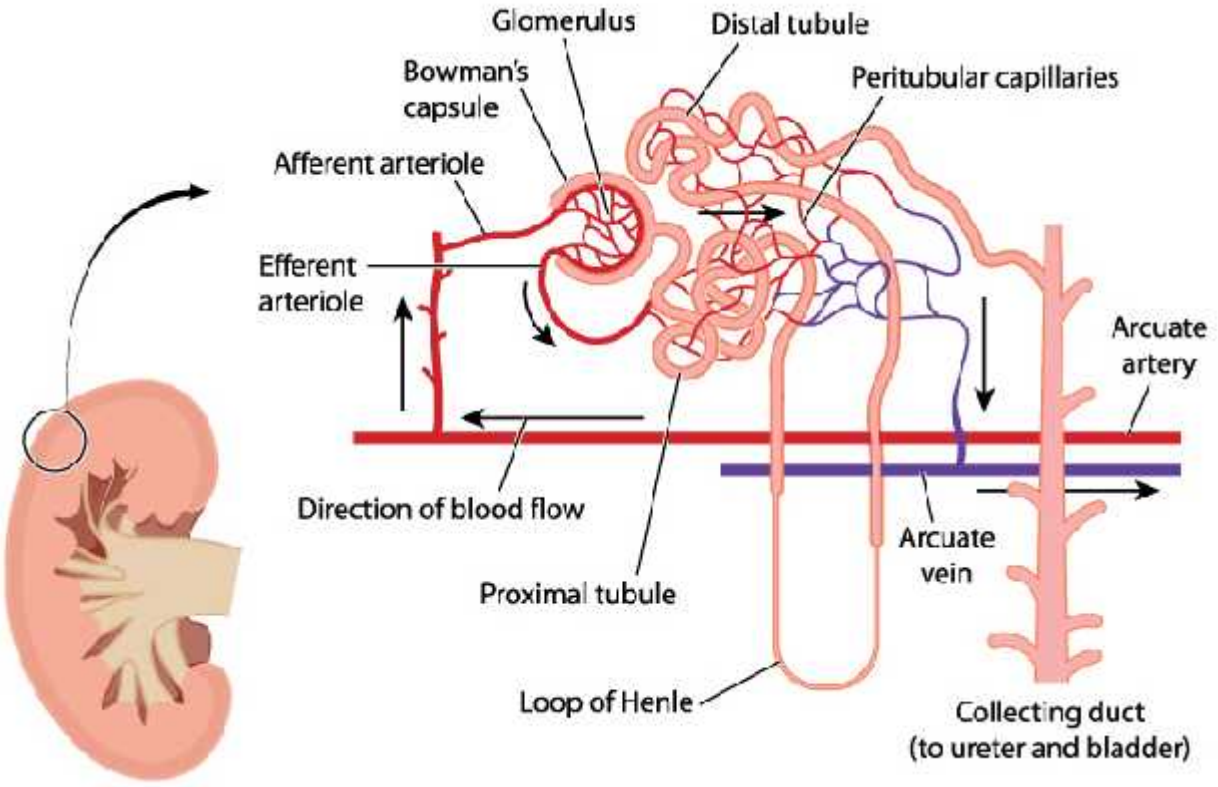
AIM OF THE STUDY

To document the

1. Clinical profile and
2. Etiological profile

of 50 randomly selected patients with clinically suspected Glomerular diseases by performing Renal biopsy in them at the Nephrology Department of Tirunelveli medical college and Hospital , Tirunelveli.

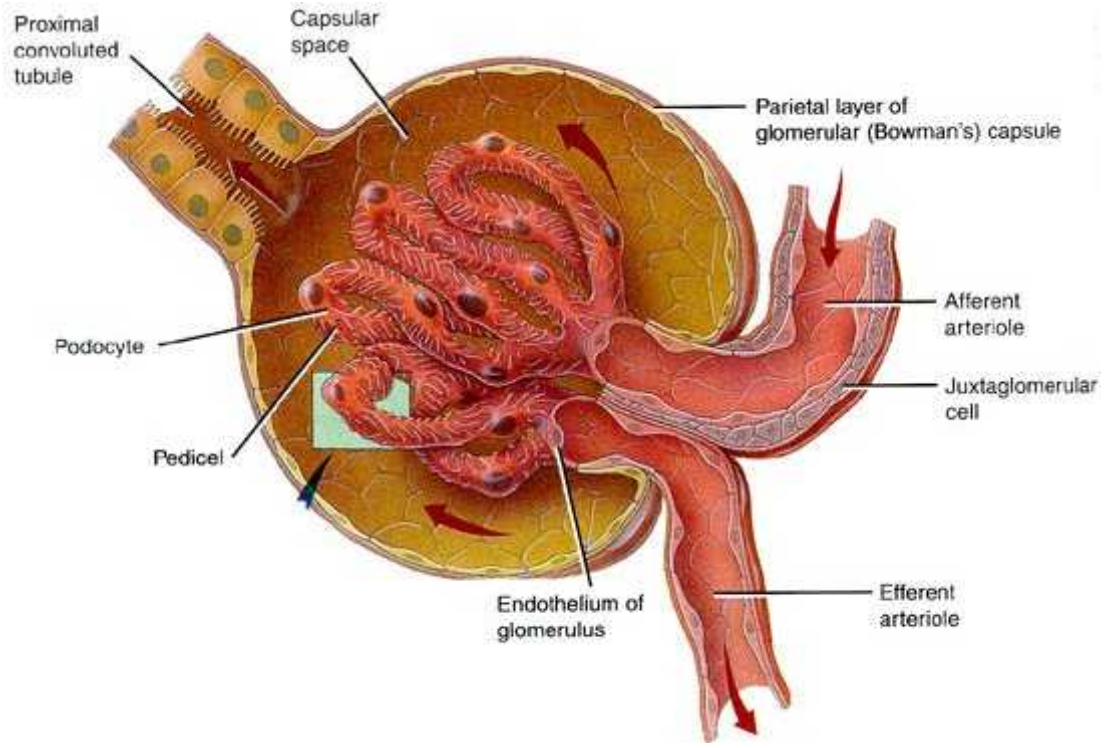
REVIEW OF LITERATURE



The renal cortex and renal medulla of each kidney contain over one million microscopic filtering structures called nephrons. ***Nephrons are the functional units of the kidney***. Each nephron is capable of filtering the blood and producing urine.

Nephron consists of two main components , the globe-shaped renal corpuscle or glomerulus and a long tube of epithelium called the renal tubule.

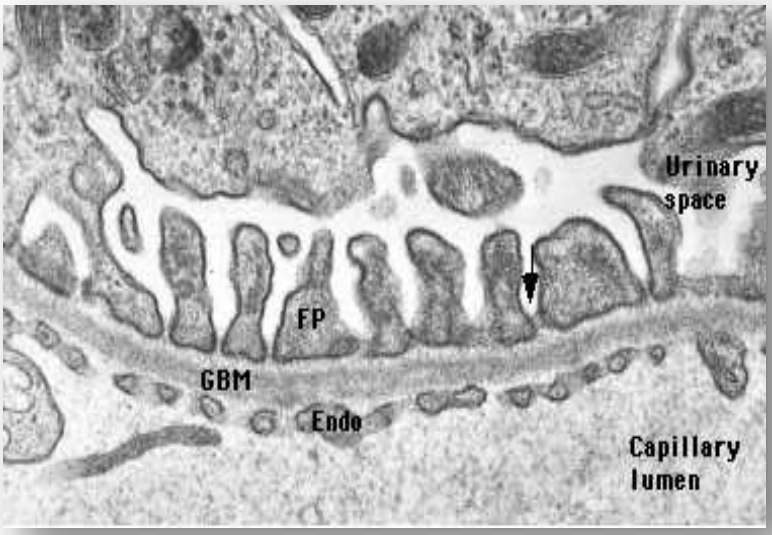
ANATOMY OF RENAL CORPUSCLE [9]



The renal corpuscle is responsible for filtering the blood. Each globe-shaped renal corpuscle consists of two parts

1. ***THE GLOMERULUS*** and
2. An outer sheath of epithelial tissue called the ***GLOMERULAR CAPSULE*** or Bowman's capsule.

The glomerulus is a group of looping fenestrated capillaries. These capillaries are called fenestrated because of the large pores, or fenestrations present within their plasma membranes and between their endothelial cells.



These fenestrations, which make the capillaries extremely “leaky,” or permeable, form a main part of the filtering structure of the renal corpuscle. Surrounding the glomerulus is the double-layered glomerular capsule, which consists of an outer parietal layer and an inner visceral layer.

The parietal layer is a globelike extension of the renal tubule consisting of simple squamous epithelium. The visceral layer consists of modified epithelial cells called podocytes that wrap around the glomerular capillaries.

Extending from each podocyte are extensions called foot processes, or pedicels. Pedicels weave together to form filtration slits, which make up another part of the renal corpuscle’s filtering structure. Between the parietal and visceral layers we find a hollow region, or lumen, called the capsular space, which is continuous with the beginning of the renal tubule lumen.

The podocytes and fenestrated glomerular capillaries form part of a complex membrane (the filtering structure we mentioned earlier) that filters blood flowing through the glomerulus. This structure allows a large volume of fluid to be filtered from the blood.

The fluid that passes through the filter to leave the glomerular capillaries, which is known as filtrate, first enters the capsular space, then flows into the renal tubule lumen.

RENAL BIOPSY [11]

In order to evaluate a kidney biopsy, the pathologist should correlate complete clinical and laboratory information with light microscope, immunofluorescence and ultrastructural findings.

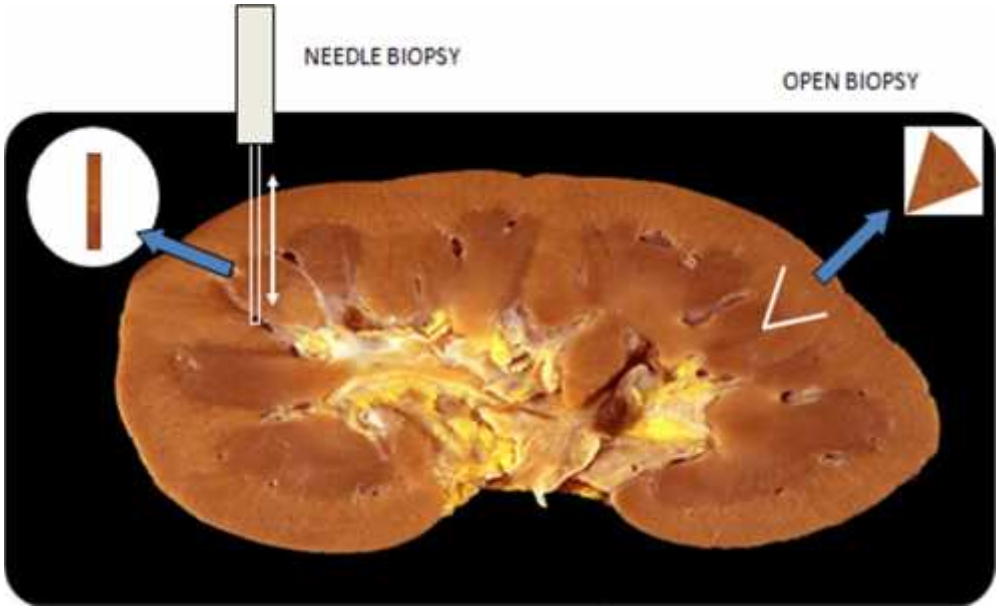
Biopsy adequacy

1-2 glomeruli : ***Electron Microscopy***

3-5 glomeruli : ***Immunofluoresence***

5-10 glomeruli : ***Light Microscopy***

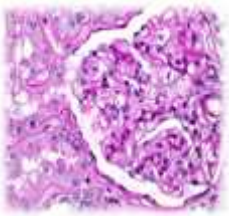
Most of the renal biopsies are done by the percutaneous route using cutting needle or by open biopsy.



STAINS FOR RENAL BIOPSY

1. **H&E** — **General**

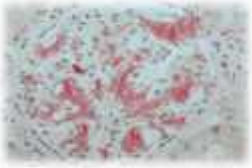
2. **PAS** — **Basement M. & Mesangial matrix**



3. **Trichrome** — **Fibrosis**

4. **Silver** — **Basement M. & Mesangial matrix**

5. **Congo red** — **Amyloid**

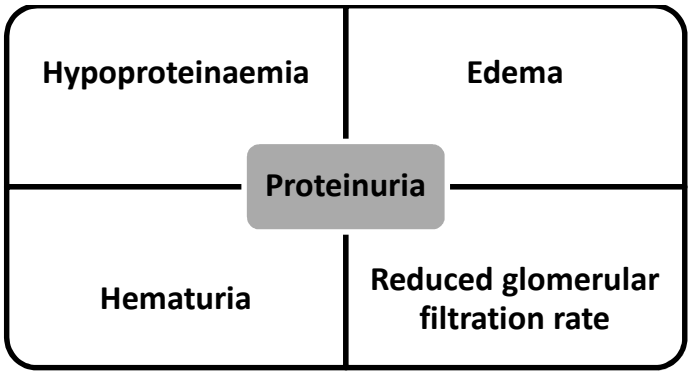


GLOMERULAR DISEASES [6]

The underlying cause of most glomerular diseases remains a mystery. Several causations have been implicated like infectious agents, autoimmunity, drugs, inherited disorders, and environmental agents .Till now the exact etiology and pathogenesis of glomerular disorders are not yet disovered completely.

Glomerular diseases may be categorized into those that primarily involve the kidney (***primary glomerular diseases***), and those in which kidney involvement is part of a systemic disorder (***secondary glomerular diseases***).

SYMPTOMS OF GLOMERULAR DISEASE [8]



PRIMARY GLOMERULOPATHIES [1]

The primary glomerulopathies are those disorders that affect glomerular structure, function, or both in the absence of a multisystem disorder. The clinical manifestations are predominately the consequence of the glomerular

lesion (such as proteinuria, hematuria, and reduced glomerular filtration rate). The combination of clinical manifestations leads to a variety of clinical syndromes.

CLASSIFICATION OF PRIMARY GLOMERULOPATHIES BASED ON CLINICAL SYNDROME [2]

Nephrotic Syndrome
Minimal change disease
Membranous glomerular nephropathy
Focal segmental glomerulosclerosis
Membranoproliferative glomerulonephritis
C1q nephropathy
Fibrillary glomerulonephritis
Acute Glomerulonephritis
Membranoproliferative glomerulonephritis
IgA nephropathy

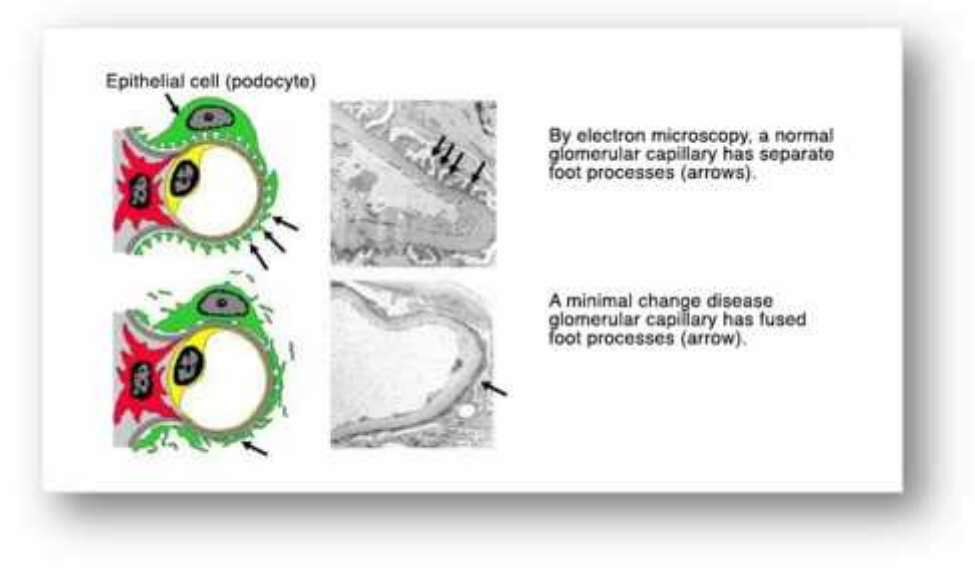
Rapidly Progressive Glomerulonephritis
Antiglomerular basement membrane disease
Immune complex crescentic glomerulonephritis
Pauci-immune crescentic glomerulonephritis
Membranoproliferative glomerulonephritis
IgA nephropathy
Membranous glomerular nephropathy (rare)
Asymptomatic Hematuria and/or Proteinuria
IgA nephropathy
Membranoproliferative glomerulonephritis

COMMON CLINICAL SYNDROMES OF PRIMARY GLOMERULAR DISEASES[2]

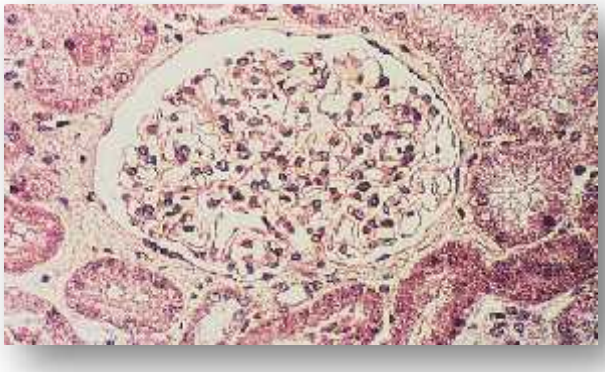
SYMPTOM	NEPHROTIC SYNDROME	ACUTE GLOMERULONEPHRITIS	RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS	ASYMPTOMATIC HEMATURIA AND/OR PROTEINURIA
Proteinuria	>3.5 g/1.73 m^2/per day*	May be in nephrotic range	May be in nephrotic range	No or non-nephrotic range
Hematuria	Variable and usually monomorphic if present	Micro- or macroscopic with RBC casts and dysmorphic RBCs	Micro- or macroscopic with RBC casts and dysmorphic RBCs	Micro- or macroscopic (may be dysmorphic with RBC casts)
Blood Pressure	Normo- or hypertension	Hypertension	Hypertension	Normotension
GFR	Variable decline, depending on diagnosis	Rapid decline (days to weeks)	Progressive decline (weeks to months)	Decline uncommon

MINIMAL CHANGE DISEASE [2]

Pathologic Definition



Accounts for 80% of all cases of the idiopathic nephrotic syndrome in children. Majority of cases seen in 3 to 4 year age groups. Male predominance of 2.5:1 in children but no difference seen in adults. 80-90% idiopathic. Associated with infectious disease, recent immunization, ingestion of heavy metals. In adults related to use of NSAIDS.



On light microscopy changes are seen in the convoluted tubules where large amounts of lipid and protein droplets accumulate in the cell cytoplasm.(lipoid nephrosis).In contrast all the glomeruli appear normal.

No deposits of complement or immunoglobulins are recognized in IF. (nil deposit disease)

Pathophysiology and Natural History

The underlying mechanism(s) leading to MCD is unknown. Some studies have implicated upregulation of various cytokine activities, including interleukin-2, during disease activity.

It has been postulated that this may induce glomerular permeability factor(s) that interfere with normal function of the charge-selective barrier to filtration of serum proteins.

Signs and Symptoms

A full blown nephrotic syndrome with heavy proteinuria often of selective type is the most common presentation.

Treatment

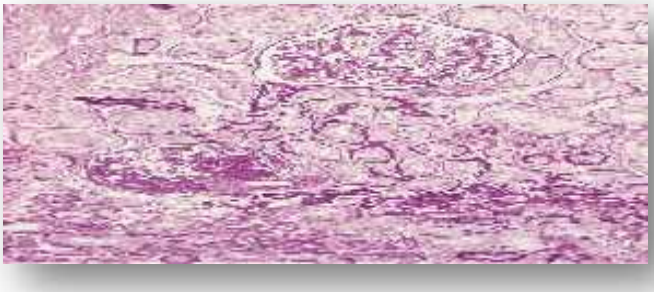
Nonimmunosuppressive management includes diuretics, ACE inhibitors, ARBs, and statins. Patients are exquisitely steroid sensitive, with a high remission rate. Relapses and steroid dependence are common and may

require cytotoxics; steroid resistance is rare and may be treated with cyclosporine.

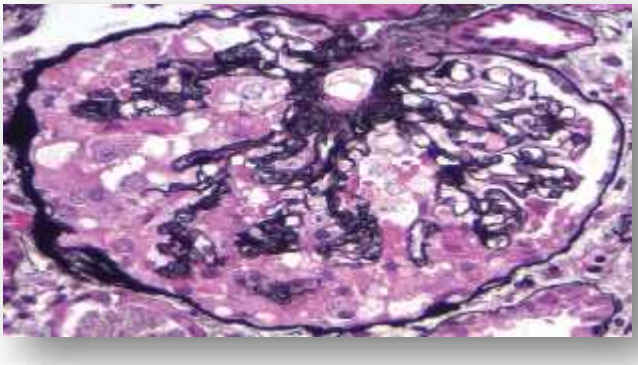
FOCAL SEGMENTAL GLOMERULOSCLEROSIS [2]

Pathologic Definition

The hallmark of kidney biopsy is an increased degree of scarring seen on light microscopy of some but not all of the glomeruli present (focal) that involves some but not all portions of the affected glomeruli.



One of the glomeruli shows segmental sclerosis while others appear unremarkable. Tubular atrophy is also seen.



Collapsing glomerulopathy. Visible retraction of the glomerular tuft, narrowing of capillary lumens, proliferation and swelling of visceral epithelial cells, and prominent accumulation of intracellular protein absorption droplets in the visceral epithelial cells.

Primary FSGS makes up approx 10% to 15% of nephrotic syndrome in children and 20% to 30% in adults. It is the predominant cause of idiopathic nephrotic syndrome in adults.

Pathophysiology and Natural History

Similar to MCD, primary FSGS is believed to occur as a result of a T-cell disorder resulting in the production of a circulating permeability factor, the identification of which has proven elusive, but may be a cytokine or lymphokine.

Signs and Symptoms

Characterized by proteinuria commonly in nephrotic range.

Treatment

Prednisone therapy is used at 1 mg/kg/day for at least 4 months.

Monitor degree of proteinuria to determine therapeutic success.

Calcineurin inhibitors are useful for those intolerant of high-dose steroids but without advanced azotemia.

MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS [2]

Pathologic Definition

Biopsy specimens from patients with membranoproliferative glomerulonephritis (MPGN) are characterized by global capillary wall thickening and glomerular hypercellularity

MPGN pathognomonic changes on electron microscopy are subendothelial and mesangial electron-dense deposits.

Pathophysiology and Natural History

Similar to MGN, MPGN is classified as an immune complex disease and the presumptive pathophysiologic mechanism is the inappropriate production of antibodies recognizing a nephritogenic antigen.

Signs and Symptoms

Approximately 50% exhibit signs and symptoms typical of the nephrotic syndrome, whereas 25% of patients only present with asymptomatic hematuria and proteinuria.

The remaining 25% present more severely, with the acute nephritic syndrome.

Treatment

Restrict immunosuppressive patients to those at highest risk for progression-heavier proteinuria, nephritic syndrome, elevated creatinine level at baseline, and crescents on biopsy.

It is crucial to exclude secondary causes, most notably hepatitis C, before initiating immunosuppressive therapy.

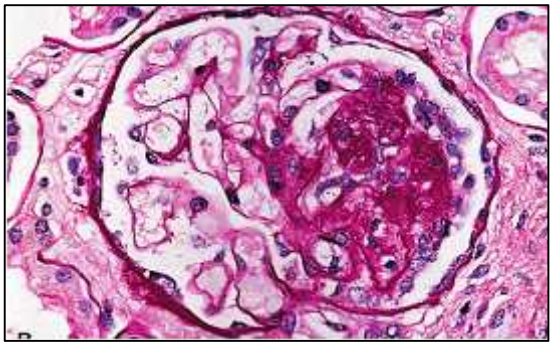
Prednisone, 2 mg/kg every other day for many months, tapered to 20 mg every other day for several years, may be given

C1Q NEPHROPATHY [3]

Pathologic Definition

Poorly understood entity .Histological features resembling lupus nephritis .C1q nephropathy falls within the clinical-pathologic spectrum of FSGS .

Light microscopy shows variable histomorphology with no significant glomerular abnormality, mesangial proliferation, diffuse proliferative glomerulonephritis. **FSGS** is the most common pattern. ‘Wire loop’ lesions are seen.



Diagnostic confirmation of C1q nephropathy is arrived at when amorphous electron dense deposits are demonstrated in the mesangium and glomerular capillary wall. Podocyte injury can also be noted

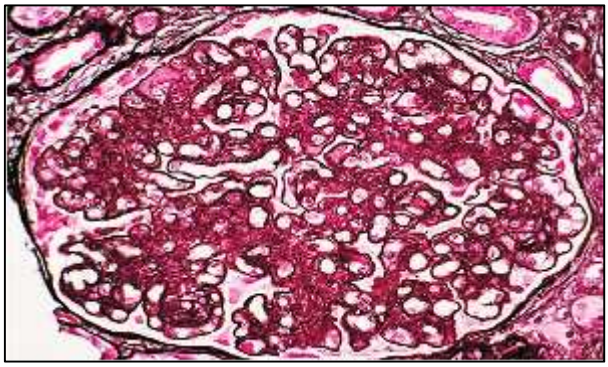
Treatment

Corticosteroids as first line treatment. Imunosuppressive therapy in steroid non responders is the treatment of choice.

FIBRILLARY GLOMERULONEPHRITIS [4]

Pathologic Definition

This glomerular deposition disease is characterized by infiltration of the glomerular capillary walls and the mesangium by eosinophilic, PAS positive, silver negative, Congo red negative and thioflavin negative material.



Signs and symptoms

The clinical presentation does not distinguish fibrillary glomerulonephritis from other primary cases of proteinuria and nephrotic syndrome. All the patients have proteinuria at the time of presentation, and more than half have nephrotic syndrome.

Hypertension, hematuria and renal insufficiency frequently accompany proteinuria. The clinical involvement is limited to the kidney, with rare exceptions, at extra-renal sites, e.g. the pancreas, liver and lungs.

Treatment

No effective therapy has been documented, although patients are often treated with corticosteroids, with or without ACE inhibitors, cyclophosphamide or other immunosuppressive drugs.

IgA NEPHROPATHY [2]

Pathologic Definition

It is the commonest form of glomerulonephritis resulting in ESRD throughout the world. Male predominance.

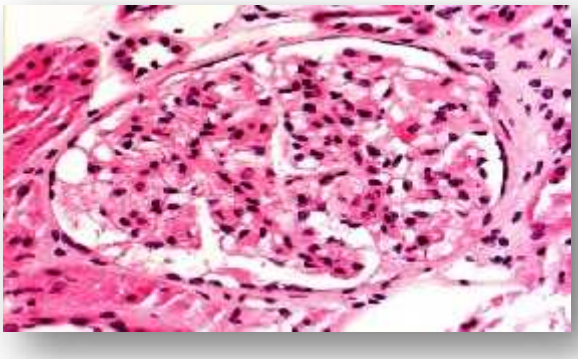
Also known as Berger's disease / Synpharyngitic glomerulonephritis

Frequent cause of gross and microscopic hematuria . Characterized by the presence of prominent IgA deposits in the mesengeal region. Suspected by light microscopy, but diagnosis is made only by immunochemical method.

Pathophysiology and Natural History

Abnormalities of immune regulation leads to increased IgA synthesis in response to respiratory or gastrointestinal exposure to environmental agents.

IgA1 (nephritogenic form) and IgA1-containing immune complexes are then trapped in the mesengium, where they activate the alternative complement pathway and initiate glomerular injury.



Glomeruli may be normal or may show mesengial widening and endocapillaryproliferation.The mesengial widening is due to cell proliferation, accumulation of matrix and immune deposits.

Signs and Symptoms

1. 0%-50% of cases : Single or sparsely recurrent episodes of gross hematuria
2. 40% of cases : Asymptomatic microscopic hematuria
3. 20% of cases : Hematuria and the nephrotic syndrome

Treatment

Treatment is dictated by clinical presentation and biopsy findings.

Moderate-risk patients should receive ACE inhibitors or ARBs, or both, statins, and possibly fish oil therapy. Severe cases are treated with steroids, with or without cyclophosphamide.

Prognosis

Generally, the long-term renal prognosis of IGAN is favorable. The most common clinical and most benign course is isolated events of macroscopic hematuria without proteinuria and normal renal function. These patients do well over time, without significant renal events.

Patients with persistent urine abnormalities, especially those with higher degrees of proteinuria, are at risk for progression of renal disease over time, although the pace may be significantly slowed by the treatment regimens discussed. The worst prognosis is limited to those with crescentic glomerulonephritis, as would be expected.

SPECIAL CASES OF CRESCENTIC GLOMERULONEPHRITIS [2]

Pathologic Definition

There are other forms of primary crescentic glomerular diseases pathologically distinct from those discussed that also lead to rapidly progressive glomerulonephritis (RPGN). Anti–glomerular basement

membrane (anti-GBM) disease refers to cases of RPGN characterized by linear staining, as opposed to granular patterns (as in MPGN or IGAN) of IgG along the glomerular basement membranes, almost always in the presence of cellular crescents and fibrinoid necrosis, but usually in the absence of significant hypercellularity.

Pauci-immune antineutrophil cytoplasmic antibody (ANCA)-associated crescentic glomerulonephritis is characterized by a necrotizing, hypercellular, crescentic, glomerular lesion similar to that in anti-GBM disease that lacks any significant immunoglobulin staining in a granular or linear pattern.

SECONDARY GLOMERULAR DISEASES [7]

In many diseases renal involvement is a part of a generalized processe.g. diabetes mellitus and systemic lupus erythematosus. Renal involvementmay be the dominant lesion or may be just an incidental finding. Generally,when the kidney is involved, the prognosis and type of treatment are changeddrastically.

SYSTEMIC LUPUS ERYTHEMATOSUS AND LUPUS NEPHRITIS

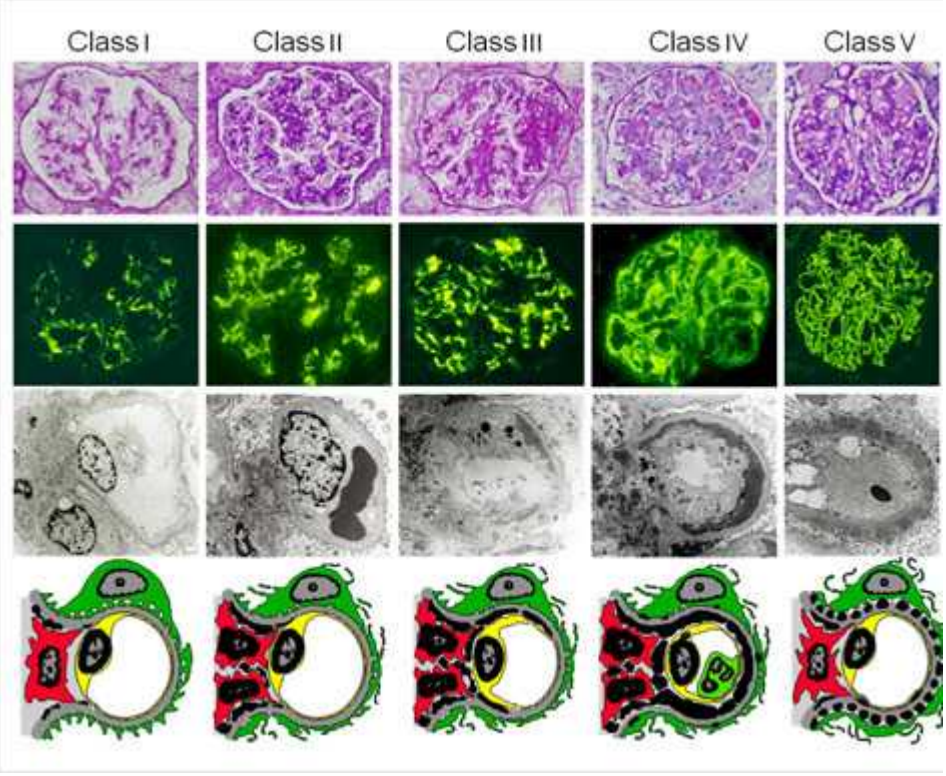
SLE is an autoimmune disease with systemic manifestations. It affects1/10,000 population. The incidence is higher in females than in males (9: 1). It affects caucasian more than black and occurs more in adolescents

than inelderly. Most probably the disease reflects an exaggerated response tocommon environmental agents in a genetically susceptible host.

Circulating and in-situ formation of DNA-anti-DNA immune complexes are thought to be the main pathogenic mechanisms for SLE. Complement deficiency may be a promoting factor.

Pathology of lupus nephritis

According to the World Health Organization (WHO), lupus nephritiscould be one of five classes:



Class I (no change) in which kidney biopsies show no changes by lightmicroscopy, few immune deposits (+) may be seen in the mesangium by I.F. and by E.M.

Class II (mild mesangial proliferative) where mild mesangial hypercellularitymay be seen by L.M. and IF and EM may show deposits in the mesangium(++) and sometimes in the subendothelial area (+).

Class III (focal and segmental proliferation), in this type, light microscopyshows evident segmental proliferation, necrosis and occasionally hyalinethrombi, IF and EM show more marked deposits in the mesangium (+++) andto less extent in the subendothelial area (+).

Class IV (diffuse proliferation), light microscopy shows diffuse hypercellularity,membranoproliferative changes, glomerular tuft necrosis, crescents, and wireloops. IF and EM show extensive deposits (+++) in all areas (mesangial,subendothelial and subepithelial).

Class V (diffuse membranous), light microscopy shows capillary wallexpansion by subepithelial deposits, with some mesangial hypercellularity. IFand EM show deposits mainly in the subepithelial (+++) area, but alsodeposits may be seen in the mesangium (++) and subendothelial area (+).

Diagnosis

For diagnosis of SLE, four or more of the criteria which have beenestablished by The American Rheumatism Association (ARA) should beencountered.

The diagnosis should be confirmed by screening for Anti-nuclearantibodies (ANA) and the more specific anti-double stranded DNA (antidsDNA). Measurement of ESR, complement component C3, C4 andCirculating Immune Complexes (CIC) may help in assessing disease activity.

The ARA criteria for diagnosis of SLE include:

1- Malar rash. 2- Discoid rash 3- Photosensitivity 4- Oral ulcers

5- Arthritis 6- Serositis7- Renal disease 8- Neurological disorders(seizures, psychosis) 9- Hematologic disorders (haemolytic anaemia, lymphopenia, leukopenia, thrombocytopenia) 10-Immunologic disorders (positive LE cell test, anti-DNA, anti-sm antibody)11-Positive anti nuclear antibody.

RENAL INVOLVEMENT IN VASCULITIS

Among different types of vasculitis, polyarteritis nodosa (PAN) and Wegener's Granulomatosis (W.G.) stand as the more common diseases affecting the kidney. Polyarteritis nodosa is either classic (involving medium sized-vessels as renal arteries with aneurysm formation) or microscopic involving small arteries and arterioles presenting with manifestation of glomerulopathies (mostly PRGN).

The classic type of polyarteritis nodosa may present with ischaemic renal changes, hypertension, immobilization with renal infarctions or haemorrhage related to the kidney (haematuria, peri-renal hematoma resulting from rupture of aneurysm). Concomitant mesenteric, coronary or cerebral vessels affection could be detected.

Wegener's granulomatosus mainly involves small vessels with early, major disease of respiratory tract excluding asthma. Granulomata are characteristic but not essential feature for diagnosis of W.G.

HENOCH-SCHÖNLEIN PURPURA (HSP)

HSP is a multisystem disease with renal, gastrointestinal andcutaneous manifestations. It usually affects children 5-15 years old with a slight preponderance of males. Full recovery is common in children. But in adults, the course could be problematic.

Renal involvement is documented in 10-30% of the cases, but in some series, it reaches up to 90% of the cases. The primary abnormality is most probably defective handling of mucosally presented antigen.

 .

Treatment and Prognosis

Generally, the disease is self-limiting. However 5-20% of cases (especially adults) may show persistence or even progression to uraemia. Signs of bad prognosis include patients with.Severe disease at presentation, persistent nephrotic syndrome, severe renal impairment and crescentic G.N.

ESSENTIAL MIXED CRYOGLOBULINAEMIA

Cryoglobulinaemia is a wide range of diseases associated with formation of cryoglobulins. While patients with cryoglobulinaemia usually present with the manifestation of the original disease, 20-30% of patients with mixed cryoglobulinaemia present with disease (vasculitis) caused by cryoglobulin itself. This is termed essential mixed cryoglobulinaemia.

PROGRESSIVE SYSTEMIC SCLEROSIS (PSS)

(Scleroderma Syndrome)

PSS is a disease characterized by progressive fibrosis of skin and internal organs of undetermined etiology. The condition may follow a long benign or short malignant course.

Renal pathology

Almost 50-100% of PSS cases show renal involvement. Interlobular arteries show narrowing of lumen and thickening of the wall with onion-skin appearance. Glomeruli usually show intracapillary fibrin deposits, mesangiolysis, rarely mesangial proliferation or crescent formation.

Treatment

The only available treatment is symptomatic. In case of hypertension, ACEls are the treatment of choice.

DIABETIC NEPHROPATHY

Microangiopathy with neuropathy, retinopathy and nephropathy are complications known to develop in the majority of long-term diabetics. Renal failure causes death in up to 40% of diabetics, being 17 times more common than in non-diabetics.

In Juvenile diabetics, nephropathy passes into 6 stages: 1- very early stage in which GFR is supernormal, 2- stage of microalbuminuria, 3- stage of

clinical proteinuria, 4- stage of nephrotic syndrome, and hypertension, 5- stage of renal impairment then, 6- stage of end stage renal failure. Stage of microalbuminuria and high GFR may continue for several years and clinical proteinuria usually settles 10-15 years later. Once clinical proteinuria is established, the disease becomes progressive to end stage renal failure.

The above described stages are the natural history in insulin dependent (type I) diabetics. In type II diabetics, the renal disease is usually well established when first discovered clinically.

Pathogenesis of diabetic nephropathy

Two mechanisms are claimed to be responsible for diabetic glomerulosclerosis. These are:

1.Hyperfiltration and hypertrophy of the renal glomeruli.

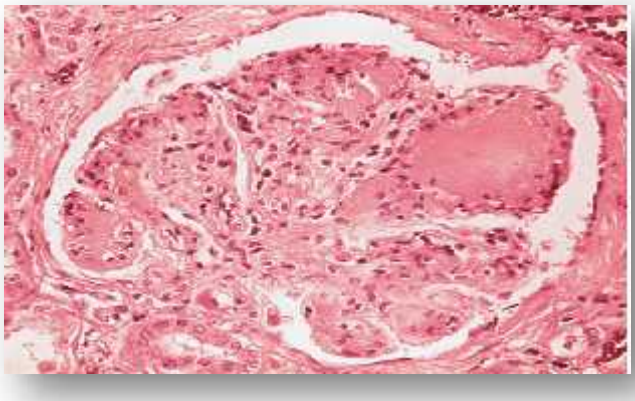
2. Glycosylation of glomerular structural proteins.

Pathology

1-progressive thickening of the GBM

2- widening of the mesangium by PASpositive material

3- focal global sclerotic lesions known as Kimmelstiel-Wilson nodules and 4- narrowing of glomerular capillaries.



ALSO KNOWN AS INTERCAPILLARY GLOMERULOSCLEROSIS OR KIMMELSTIEL-WILSON DISEASE.

Other distinctive lesions which may be seen in kidney biopsies of diabetic patients are fibrin caps, capsular drop lesions and gross hyalinization of arterioles. Also, interstitial scarring infiltarion and tubular atrophy are seen.

HEREDITARY GLOMERULOPATHIES

ALPORT SYNDROME

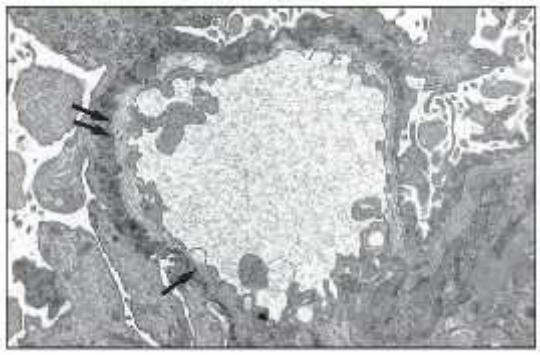
Alport Syndrome is an autosomal dominant inherited disease with variable penetrance, sometimes with X-linkage. Clinically, the patients show combination of renal disease, nerve deafness ocular defects (anterior Lenticonus, cataract, macular lesions) and platelet defect (macrothrombocytopathic thrombocytopenia).

The basic defect is in the type IV collagen which is normally present in the GBM, lens and cochlea.

Pathology

The characteristic feature of Alport's syndrome is seen in kidney

sections examined by E.M. which are lamellation, splitting and thinning of the GBM. As the disease progresses the GBM takes the form described as "basket weave" appearance



Clinical features

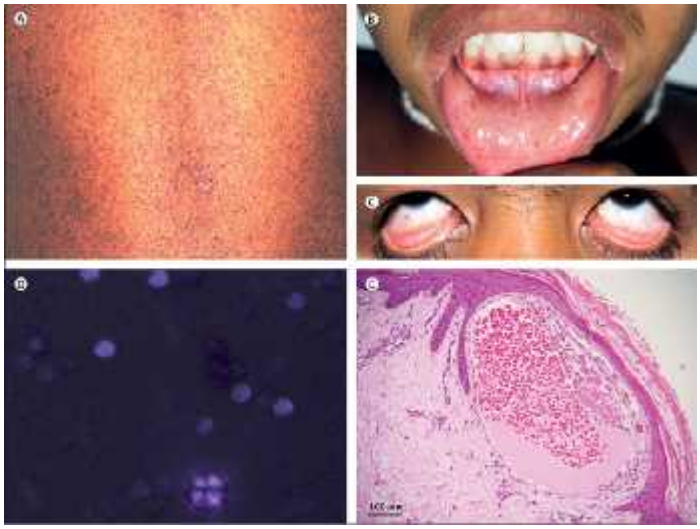
Haematuria is the main feature of this disease. It is microscopic an may be detected even at birth. Later it becomes macroscopic with intercurrent illness

presents in the transplanted kidney. If this occurs, the patient may need plasma exchange sessions.

FABRY'S DISEASE

Fabry's disease results from the deficiency of the enzyme agalactosidase. This, in turn, results in an accumulation in all tissues of glycosphingo-lipids, cerebroside dihexoside and cerebroside trihexoside. The

disease is inherited as X-linked, the homozygous males are severely affected while the heterozygous females are asymptomatic.



Clinical Features

1. Skin lesions in the form of angiokeratomas which are red papules in the mouth, lower abdomen, buttocks and pubic region Neurologic manifestations in the form of periodic episodes of severe pain due to involvement of dorsal root ganglia.
2. Cardiac manifestations as hypotension and ischaemic heart disease.
3. Renal manifestations include, haematuria, proteinuria and progressive uraemia. Kidney sections will show changes in visceral glomerular epithelial cells, endothelial cells and tubular cells in the form of fat accumulations as seen by light microscopy and myelin as seenby EM. Usually patients die from cardiac or renal disease in fourth of fifth decades of life.

4. Screening of family members for a-galactosidase deficiency in serum, leucocytes, hair follicles and biopsy specimens is mandatory.

Treatment

Is mainly supportive, dialysis is tolerable and transplantation-inspite ofbeing successful-does not provide the missing enzyme

NAIL-PATELLA SYNDROME

(Hereditary onycho-Osteo-dysplasia)

This is characterized by a generalized disturbance in collagensynthesis leading to dysplasia of nails, skeletal deformities (especiallyhypoplastic displaced patella, deformed elbow, iliac horns, scoliosis) and renalinvolvement.

The disease is transmitted as autosomal dominant trait. Renal manifestations include haematuria and proteinuria, but rarely nephroticsyndrome or renal failure occurs.

Histopathologically, there is an irregular thickening of the GBM withnumerous lucent areas containing electron dense fibrils.

BACTERIAL ENDOCARDITIS

In bacterial endocarditis occurring in patients with rheumatic valvedisease and in intravenous drug abusers the incidence of glomerulonephritisis high.

Pathogenesis and Pathology

Renal involvement in endocarditis is mainly immunologic. It is a sort of immune complex mediated glomerular damage. The immune complexes arefound to contain bacterial antigen, antibodies and complement.

Clinical features and diagnosis

The clinical features of renal involvement in endocarditis may vary fromasymptomatic urine abnormalities especially in the focal and segmental lesions to severe RPGN as in the diffuse proliferative lesions.

In patients with bacterial endocarditis renal impairment could be due to glomerulonephritis, drug induced toxicity or secondary to cardiac failure.Tests for CIC are positive with transient hypocomplementaemia andsometimes positive ANA and rheumatoid factor.

Treatment

Treatment is that of endocarditis and symptomatic treatment for renaldisease (e.g. dialysis for renal failure).Use of steroids and immunosuppressives is rarely needed.

TUBULOINTERSTITIAL NEPHRITIS

Usually occurs in patients suffering from high fever, hypovolaemia,hemolytic jaundice, intravascular coagulation, hyperviscosity and heavy parasitaemia. There is acute tubular necrosis (toxic and ischaemic),

obstruction of distal nephron with casts and interstitial infiltration withmononuclear cells.

Peritubular capillaries are congested with erythrocytes,macrophages laden with malarial pigment and mono-nuclear cells.Renal failure in falciparum malaria is usually hypercatabolic with rapidrise in blood urea and serum creatinine. Hyperkalaemia and hyperuricaemiaoccur particularly with intravascular haemolysis.Haemodialysis is preferable to peritoneal dialysis when dialysis supportis indicated. Exchange transfusion is indicated with heavy parasitaemia.

GLOMERULOPATHY SECONDARY TO VIRUS INFECTION

A variety of viral infections may be associated with features of acuteglomerulonephritis. However, it is usually milder than it is in post streptococcal glomerulonephritis.

Classification

(1) Herpes virus: - cytomegalovirus , Epestien Bar virus.

(2) Paramyxovirus: - measles , mumps

(3) Parovirus

(4) Hepatitis viruses: - hepatitis B ,hepatitis C

(5) Retroviruses: - human immunodeficiency virus.

(6) Influenza viruses: - Influenza A & B

Mechanism of Renal affection in viral infection

(1) Direct cytopathic effect of the virus on the glomerular cells.

(2) Immune complex mediated which is due to stimulation of antibod response.

(3) Direct effect on T-cells.

PREVIOUS STUDIES

STUDY :1

ST Ramesh Chandra et all conducted a study on the " **EPIDEMIOLOGY OF BIOPSY PROVEN RENAL DISEASE (BPRD) AND TO LOOK FOR ANY CHANGING TRENDS IN RENAL DISEASE"** at **Andhra Medical College, Visakhapatnam, India**. Study population included a total of **624 cases** Patient's age ranged from 4 months to 68 years with male: female ratio 1.07:1.

According to that study the most common clinical indication for renal biopsy was ***Nephrotic syndrome*** (39%).Out of all, primary glomerular disease (PGD) was the most common BPRD, accounting for 60.25% of the total cases. ***Minimal change disease*** (20.47%) was the commonest PGD followed by focal segmental glomeruloscleros is (18.35%) and membranous nephropathy (16.22%). ***Tubulointerstitial nephritis*** (TIN) (16.66% total

BPRD) is the second most common BPRD. Acute TIN accounts for 56.73% of the total TIN.

Secondary glomerular disease (SGD) is third most common. SGD accounts for 13.78% of total BPRD. ***Lupus nephritis*** (62.79% of total SGD) was the commonest SGD followed by ***HUS/TTP*** (12.76%total SGD). ***Diabetic nephropathy***(10.46% total SGD) is third most common SGD. Chronic renal parenchymal changes (5.12% total BPRD) and vascular disease (4.17% total BPRD)were less common.

STUDY :2

Fu-de Zhou et all conducted a study about the "**THE RENAL HISTOPATHOLOGICAL SPECTRUM OF PATIENTS WITH NEPHROTIC SYNDROME"** at **Peking University First Hospital, Institute of Nephrology, Peking University**. One thousand five hundred and twenty-three **(1523)** consecutive patients (>=14 years old at renal biopsy) with nephrotic syndrome were recruited. Patients were divided into four groups according to age at the time of renal biopsy.

The renal histopathological spectrum was also compared between nephrotic range proteinuria patients with and without hypoalbuminaemia.

Among the 1523 patients, the most common cause of nephrotic syndrome was ***idiopathic membranous nephropathy*** (IMN) (20.7%),

followed by ***minimal change disease*** (MCD) (20.4%). Among the patients aged 14–24, 25–44, 45–59 and >=60 years, the most common cause of nephrotic syndrome was MCD (33.0%), lupus nephritis (LN) (23.0%), IMN (37.9%) and IMN (42.3%), respectively. Among the ***female patients*** aged 14–24 and 25–44 years, ***LN*** was the leading cause of nephrotic syndrome (35.8 and 36.2%, respectively).

The proportion of patients with renal ***amyloidosis increased in parallel with patient age***. The comparison between nephrotic patients with and without hypoalbuminaemia suggests that patients with MCD, LN or renal amyloidosis were more likely to develop

STUDY :3

U.D, K. V. Dakshinamurty, and A. Prayaga studied the **"PATTERN OF BIOPSY-PROVEN RENAL DISEASE"** at **Nizam's Institute of Medical Sciences, Punjagutta, Hyderabad.** The study population was **1849** patients. Among them, 1091 were males and 758 were females. The mean age of patients was 32.27 ± 18.37 (range 10-80) years.

The most common indication for renal biopsy was **NS**: 906 (49%), followed by **CRF**: 251 (13.6%), **RPRF**: 221 (12%), **ANS**:167 (9%), **AUA**:167 (9%), **ARF**:120 (6.5%) and **gross hematuria**:17 (0.9%). **PGN** remained the most common and important kidney disease in the patients and

accounted for 1278 (69.1%) of the total patients. Among the PGN cases, ***MCD*** (21.8%) was the leading category, followed by ***FSGS*** (15.3%), **MN** (10%), ***chronic glomerular nephritis CGN*** (9.7%), ***PIGN*** (8.1%), ***MesPGN*** (7.5%), ***DPGN*** (6.7%), ***CresGN (***6.5%), ***IgAN*** (6.3%), ***MPGN*** (5.7%) and ***focal proliferative glomerular nephritis [FPGN]*** (1.6%). IgMN (0.5%) was very rare. The diagnosis of IgMN was made after ruling out MCD and FSGS.

The most common SGN was ***LN*** (80.1%), followed by ***amyloidosis*** (8%) and ***DN*** (6.5%). TIN, VN and ESRD changes were less common diagnostic categories. There were no hereditary glomerular diseases in this analysis.

STUDY :4

Dawood Al Riyami et all conducted a study on **"THE SPECTRUM OF GLOMERULAR DISEASES ON RENAL BIOPSY" at Sultan Qaboos University Hospital, Muscat, Al-Khoud, Sultanate of Oman**.

A retrospective review of ***190 adult*** native renal biopsy reports from the pathology registry of renal biopsy performed at the hospital between ***1992 and 2010*** was taken for the study.

According to the study ***Lupus nephritis*** was the most common pathology 48/133 (36.1%) with a **female preponderance**. The most common primary glomerular disease was ***focal segmental glomerulosclerosis*** (FSGS)

26/133(19.5%), followed by ***membranous glemerulopathy (MGN)*** 13/133 (9.8%), and ***mesangial proliferative glomerulonephritis*** 6/133 (4.5%).

IgA nephropathy and acute proliferative glomerulonephritis each accounted for 4/133 (3.0%). ***Membranoproliferative glomerulonephritis*** accounted for 3/133 (2.3%). Focal proliferative and cresentic glomerulonephritis each accounted for 2/133 (1.5%). Vasculitis was not common and there ***was no report of anti-GBM disease***

STUDY :5

Jae Hyun Chang et all conducted a study on the **"CHANGING PREVALENCE OF GLOMERULAR DISEASES IN KOREAN ADULTS**" at **Yonsei University College of Medicine, Seoul, Korea**.

Patients aged 16 years or older who underwenta renal biopsy at Severance Hospital in the Yonsei University Health System from **1987 to 2006** were enrolled. All medical records were reviewed retrospectively. In total, **1818** patients (M:F = 1.02:1) were reviewed.

According to the study ***Glomerulonephritis (GN***) comprised 85.9% of the total biopsied cases. The most common primary GN was ***IgA nephropathy (IgAN)*** (28.3%), which was followed by ***minimal change disease (MCD)*** (15.5%), ***membranous nephropathy (MN***) (12.3%), focal segmental glomerulosclerosis (FSGS) (5.6%) and membranoproliferative GN (MPGN) (4.0%). The most common secondary GN was ***lupus nephritis*** (8.7%).

MATERIALS AND METHODS

MATERIALS

50 randomly selected patients with clinically suspected **Glomerular diseases** in whom **renal biopsy** was performed at the Nephrology Department of Tirunelveli medical college and Hospital, Tirunelveli.

DURATION OF THE STUDY : 1 Year

TYPE OF STUDY : Descriptive Study

SAMPLE SIZE : 50

INCLUSION CRITERIA

1. > 15 years of age
2. Proteinuria >500mg/24 hrs
3. Hematuria (Microscopic/gross)

EXCLUSION CRITERIA

1. <15 years of age
2. Any coagulopathies

METHODOLOGY

Informed consent was obtained for taking part in this study. A prospective study was conducted on patients of either sex , age >15 years who presented to the Medicine department of Tirunelveli Medical college and Hospital with proteinuria or hematuria. Patients with these features were clinically evaluated and subjected to tests like serum total protein , urine PCR , Lipid profile , BUN and renal imaging. If results were suggestive of glomerular disease percutaneous renal biopsy was done to characterize the exact pathology. 50 cases were randomly selected and the clinical and laboratory profile of all the cases were studied and the results were analysed statistically.

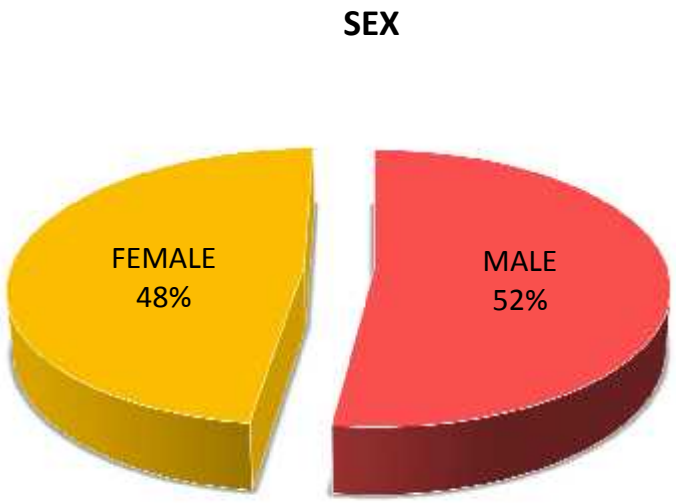
STATISTICAL ANALYSIS

Statistical analysis was done using simple percentage analysis.

OBSERVATION AND RESULTS

SEX DISTRIBUTION

	SEX	PERCENTAGE
MALE	**26**	**52%**
FEMALE	**24**	**48%**

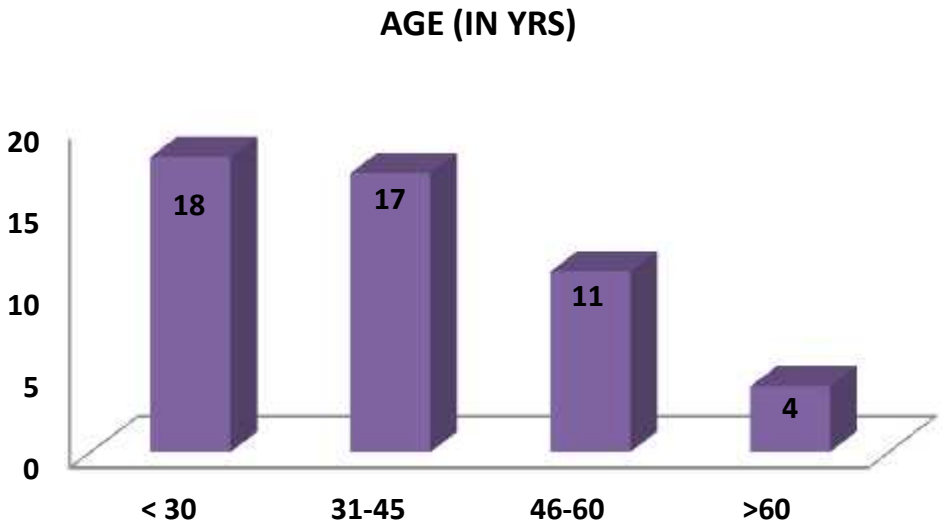


MALE: FEMALE RATIO = 1.08:1

In this study the number of males (52%) and the number of females (48%) were nearly equal.

AGE DISTRIBUTION

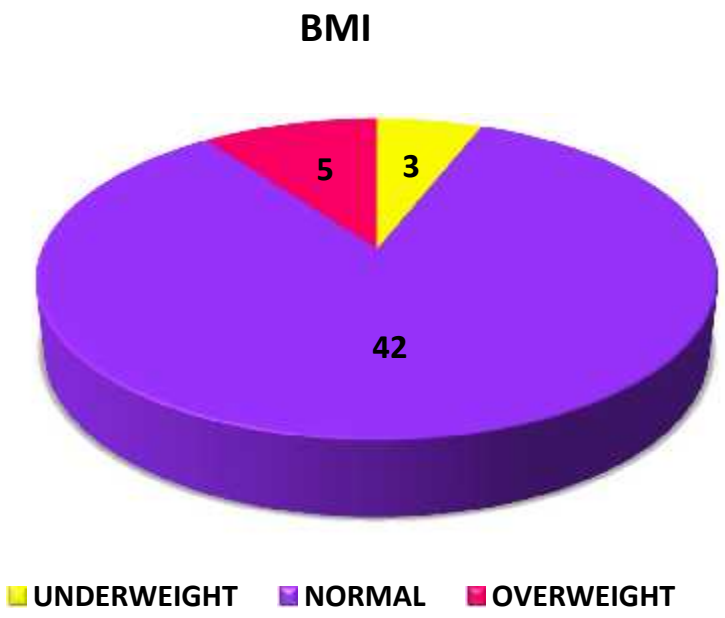
	AGE(IN YRS)	PERCENTAGE
< 30	18	36%
31-45	17	34%
46-60	11	22%
>60	4	8%



In thus study people under the age of 30 years was maximum (36%) followed by the age group of 31 to 40 years (34%). People between the age group of 46 to 60 years was 22% and people greater than 60 years was the lowest in number (4%).

BODY MASS INDEX

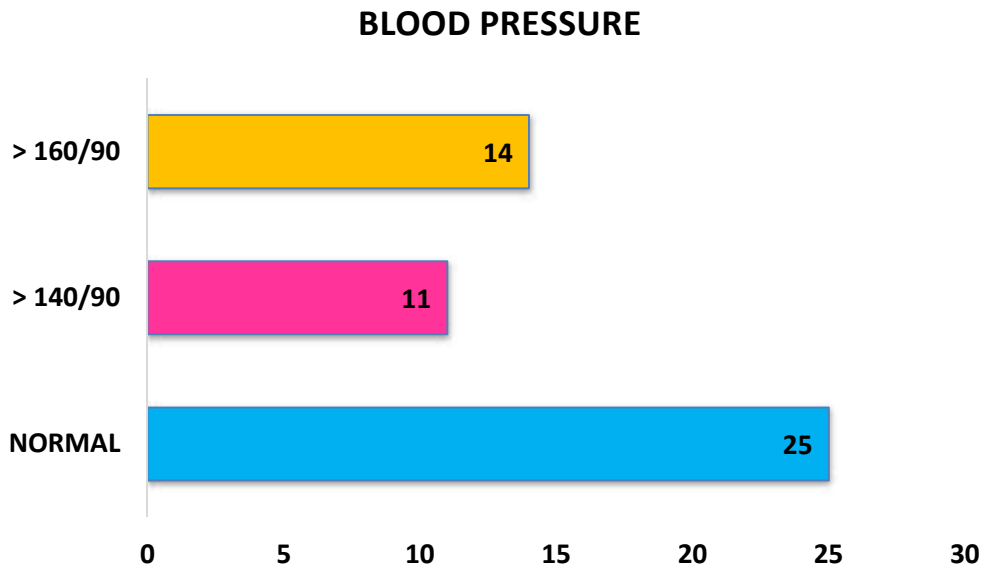
	BMI	PERCENTAGE
UNDERWEIGHT	3	6%
NORMAL	42	84%
OVERWEIGHT	5	10%



In this study 6% of people are underweight , 84% have normal BMI and the remaining 10% are overweight.

BLOOD PRESSURE

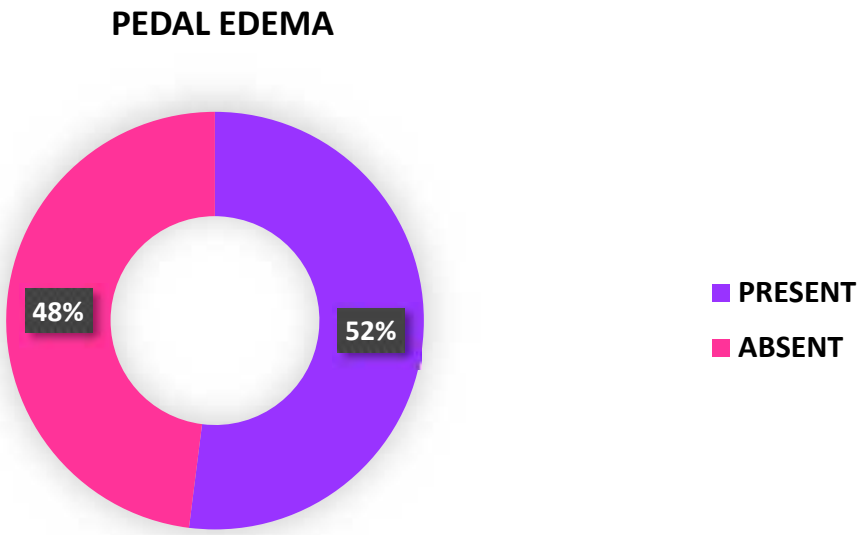
	BLOOD PRESSURE	PERCENTAGE
NORMAL	25	50%
> 140/90	11	22%
> 160/90	14	28%



In our study about 50% of people have normal blood pressure. About 22% of people have BP >140/90 mmHg. About 28% of people have BP > 160/90 mmHg.

RENAL MANIFESTATIONS

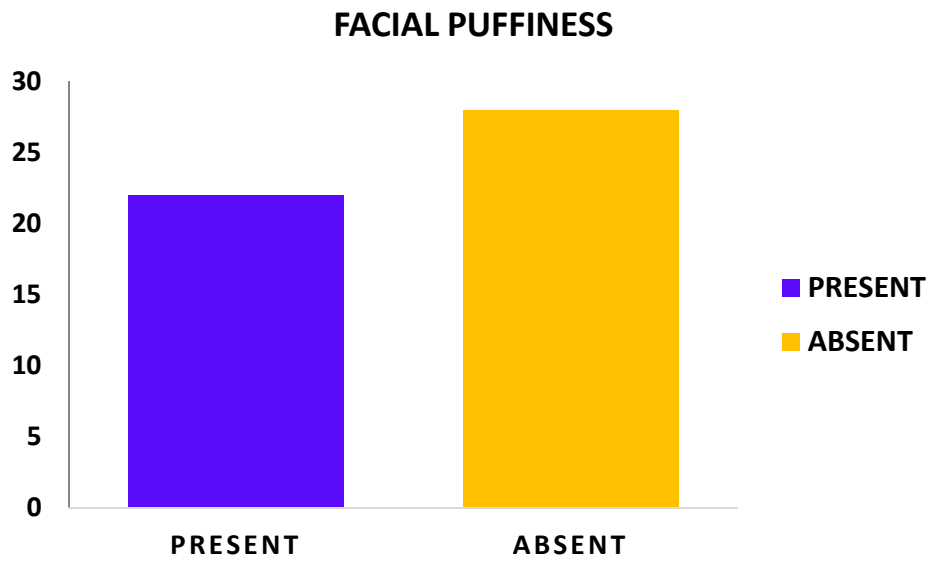
	PEDAL EDEMA	PERCENTAGE
PRESENT	**26**	**52%**
ABSENT	**24**	**48%**



In this study about 48% of people presented with pedal edema and the remaining 52% did not have pedal edema.

FACIAL PUFFINESS

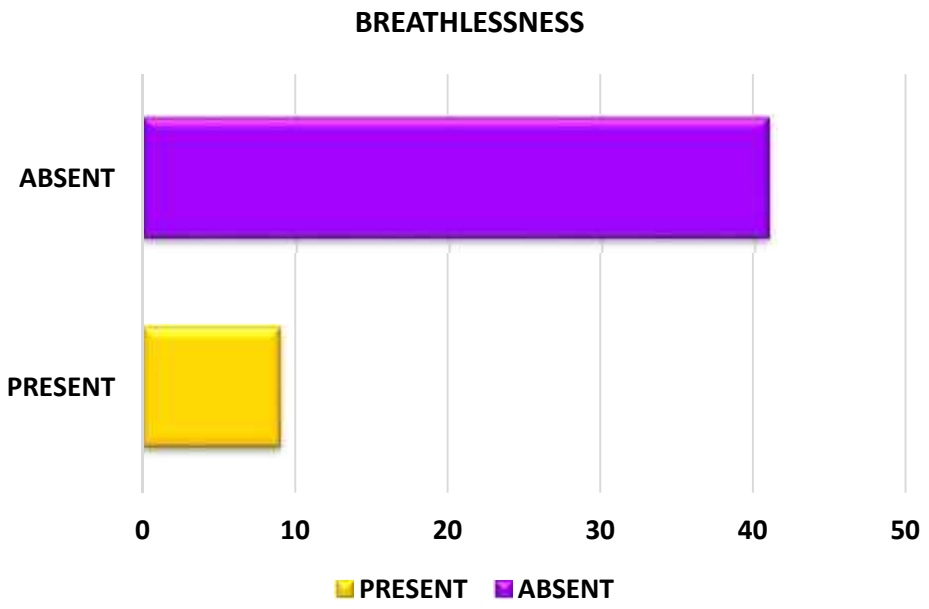
	FACIAL PUFFINESS	PERCENTAGE
PRESENT	**22**	**44%**
ABSENT	**28**	**56%**



In our study about 44% of people presented with facial puffiness and in the remaining 56% facial puffiness was absent.

BREATHLESSNESS

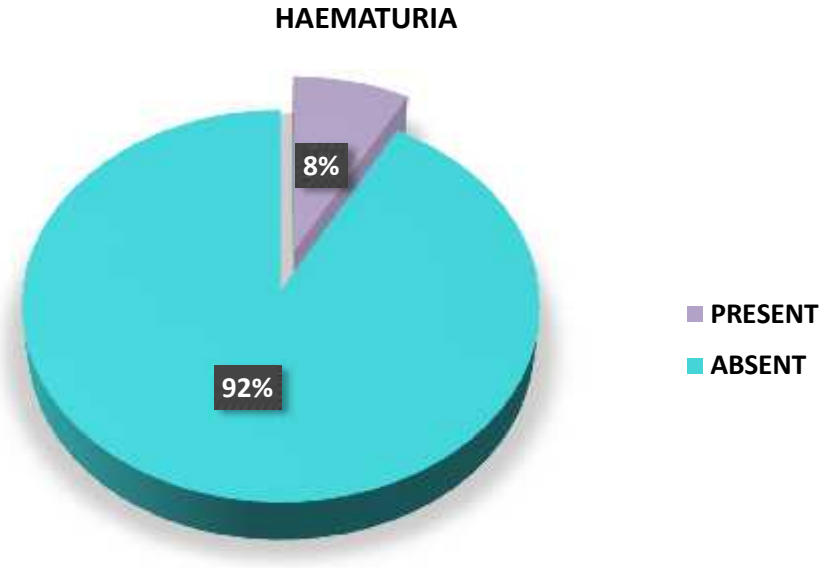
	BREATHLESSNESS	PERCENTAGE
PRESENT	**9**	**18%**
ABSENT	**41**	**82%**



In our study there was no breathlessness in 82% of individuals at the time of presentation while18% of individuals presented with breathlessness.

HEMATURIA

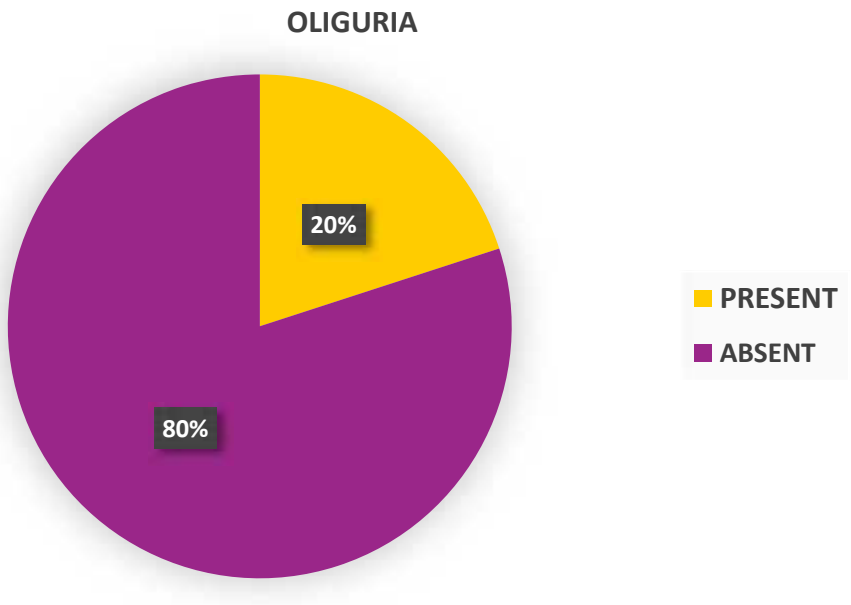
	HAEMATURIA	PERCENTAGE
PRESENT	4	8%
ABSENT	46	92%



In our study in 92% of people hematuria was absent while remaining 8% had

OLIGURIA

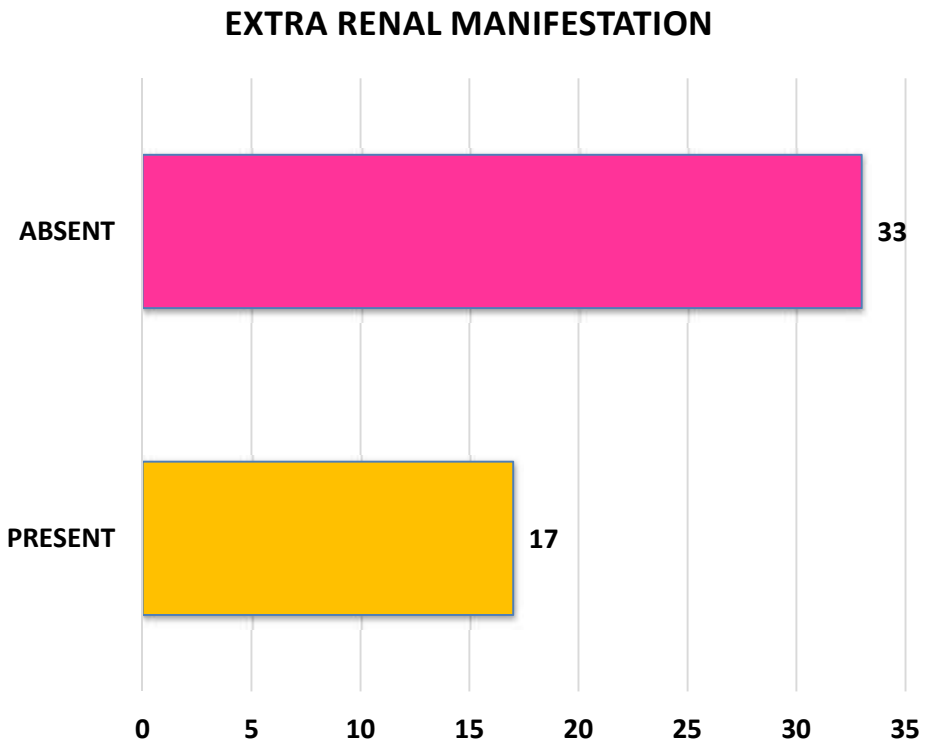
	OLIGURIA	PERCENTAGE
PRESENT	**10**	**20%**
ABSENT	**40**	**80%**



In our study 20% of individuals presented with oliguria while remaining 20% had no hematuria.

EXTRA RENAL MANIFESTATION

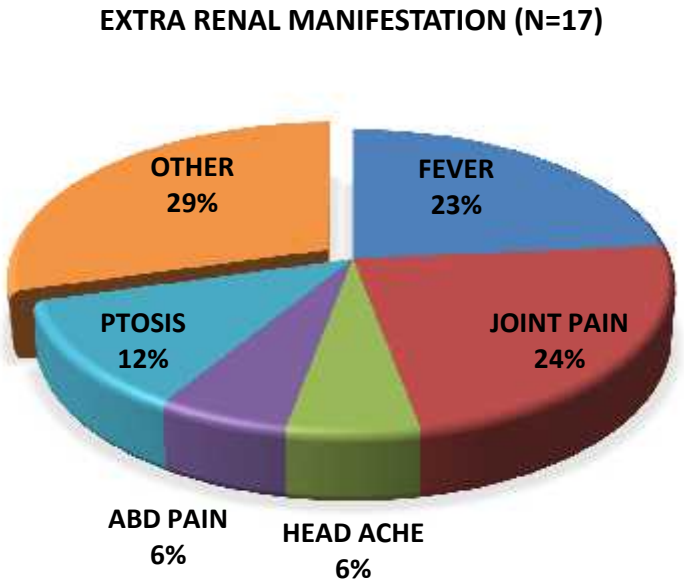
	EXTRA RENAL MANIFESTATION	PERCENTAGE
PRESENT	**17**	**34%**
ABSENT	**33**	**66%**



In our study 66% of individuals had no extra renal manifestations while 34% of individuals hadextrarenal manifestations.

EXTRA RENAL MANIFESTATION

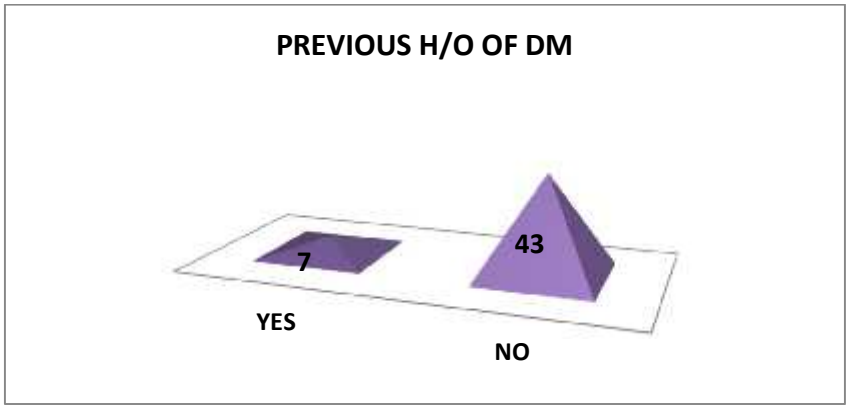
	EXTRA RENAL MANIFESTATION(N=17)	PERCENTAGE
FEVER	**4**	**23.50%**
JOINT PAIN	**4**	**23.50%**
HEAD ACHE	**1**	**5.90%**
ABD PAIN	**1**	**5.90%**
PTOSIS	**2**	**11.80%**
OTHER	**5**	**29.40%**



In our study joint pain(23%) and fever(24%) were the most common extra renal manifestations followed by ptosis (12%) , abdominal pain and headache (6%)

PREVIOUS HISTORY OF DM

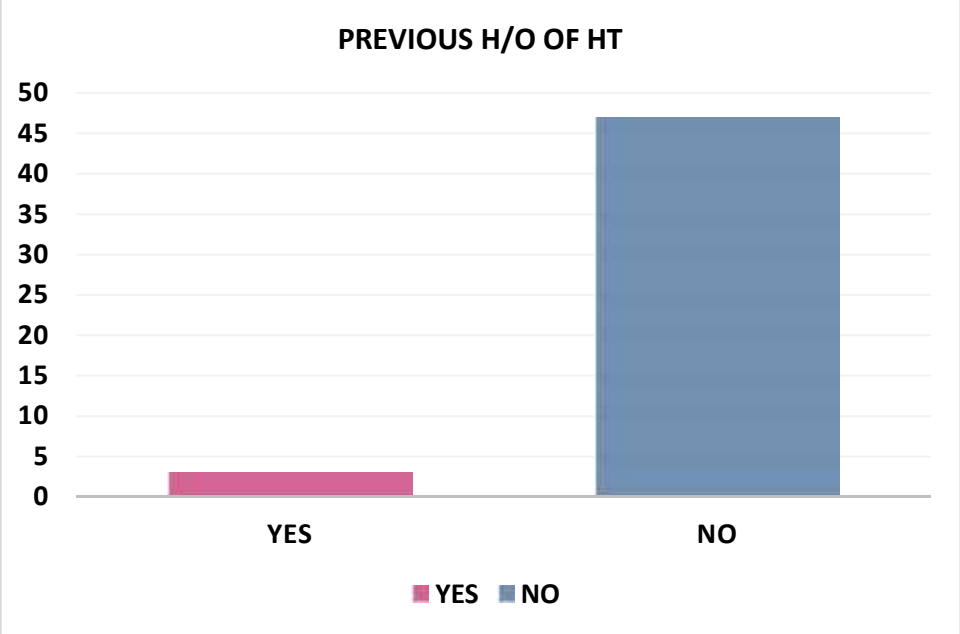
	PREVIOUS H/O OF DM	PERCENTAGE
YES	7	14%
NO	43	86%



In our study 86% of individuals had no previous history of diabetes mellitus while remaining 14% had previous history of diabetes.

PREVIOUS HISTORY OF HYPERTENSION

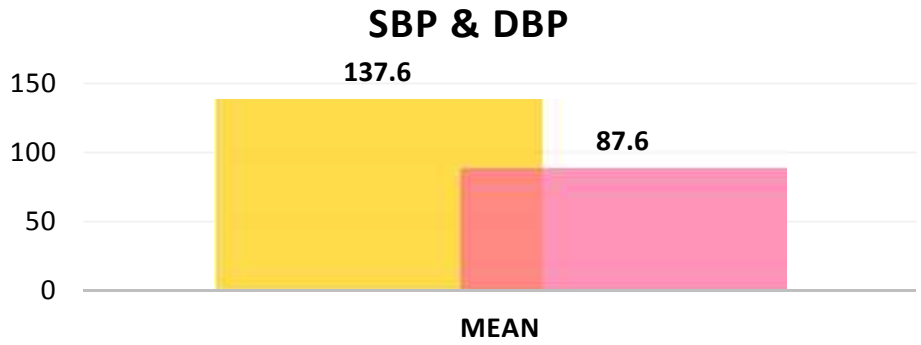
	PREVIOUS H/O OF HT	PERCENTAGE
YES	3	6%
NO	47	94%



In our study 94% of people had no previous history of hypertension while remaining 6% had previous history of hypertension

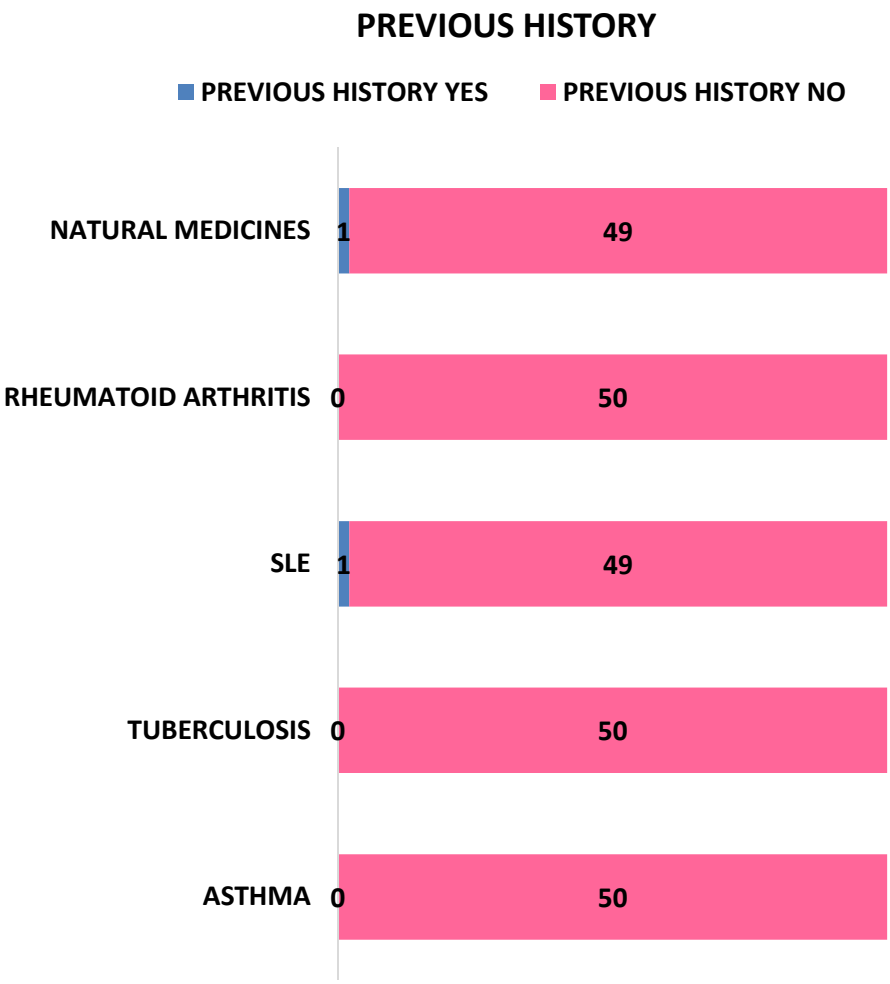
BLOOD PRESSURE

	MEAN	STANDARD DEVIATION
SYSTOLIC BP	137.6	21.24
DIASTOLIC BP	87.6	12.7



PREVIOUS HISTORY

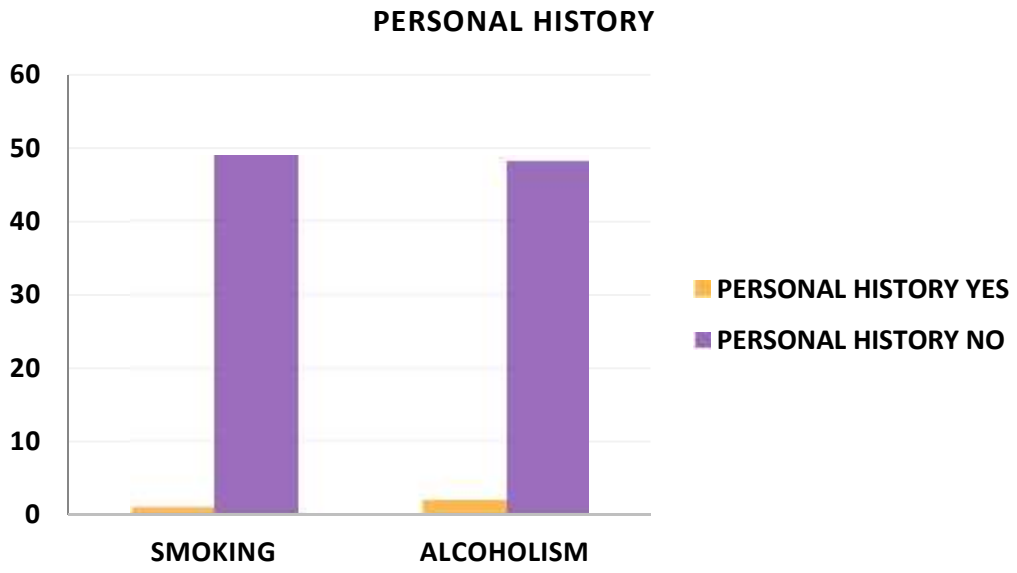
	PREVIOUS HISTORY	
	YES	**NO**
ASTHMA	**0**	**50**
TUBERCULOSIS	**0**	**50**
SLE	**1**	**49**
RHEUMATOID ARTHRITIS	**0**	**50**
NATURAL MEDICINES	**1**	**49**



In our study no individuals have previous history of rheumatoid arthritis, tuberculosis or asthma. While one individual each have previous history of SLE and have previous history of using natural medicines.

PERSONAL HISTORY

	PERSONAL HISTORY	
	YES	**NO**
SMOKING	**1**	**49**
ALCOHOLISM	**2**	**48**



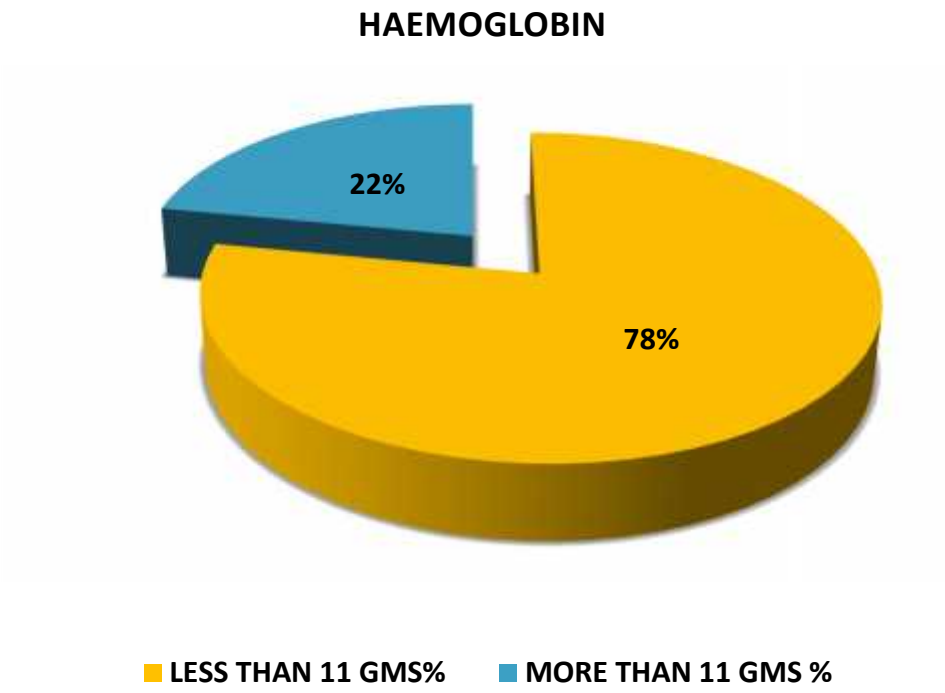
In our study only 2 individuals were alcoholic and only 1 individual was a smoker.

INVESTIGATIONS

	MEAN	STANDARD DEVIATION
RANDOM BLOOD SUGAR	132.38	35.34
TOTAL COUNT	10642	6743
NEUTROPHIL	69.42	14.96
LEUCOCYTE	25.06	12.51
EOSINOPHIL	5.12	4.82
ESR	59.46	35.91
HB%	9.88	2.38
PLATELET COUNT	288000	119000

HAEMOGLOBIN

	HAEMOGLOBIN	PERCENTAGE
LESS THAN 11 GMS%	39	78%
MORE THAN 11 GMS %	11	22%



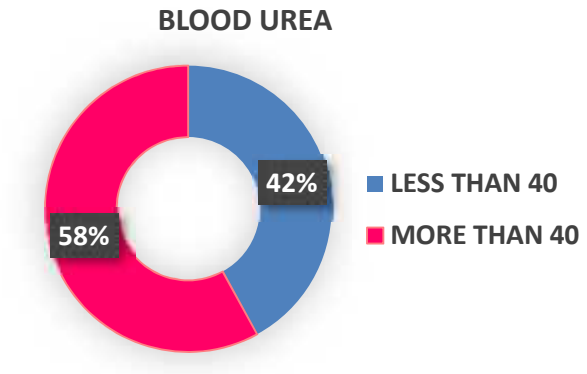
In our study 78% of individuals had haemoglobin less than 11gm%. Only 22% of individuals had haemoglobin more than 11gm %

RENAL FUNCTION TEST

	RENAL FUNCTION TEST	
	MEAN	**STANDARD DEVIATION**
UREA	**56.2**	**39.21**
CREATNINE	**2.45**	**2.14**
EGFR	**55.7**	**41.13**

BLOOD UREA

	BLOOD UREA	PERCENTAGE
LESS THAN 40	**21**	**42%**
MORE THAN 40	**29**	**58%**

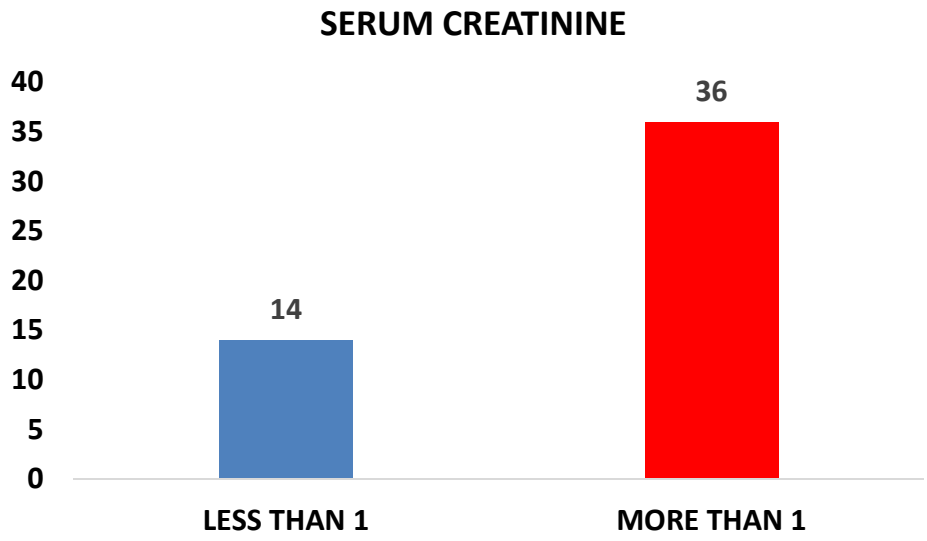


In our study blood urea levels were less than 40mg/dl in 42% of individuals.

More than 40mg/dl in 58% of individuals.

SERUM CREATININE

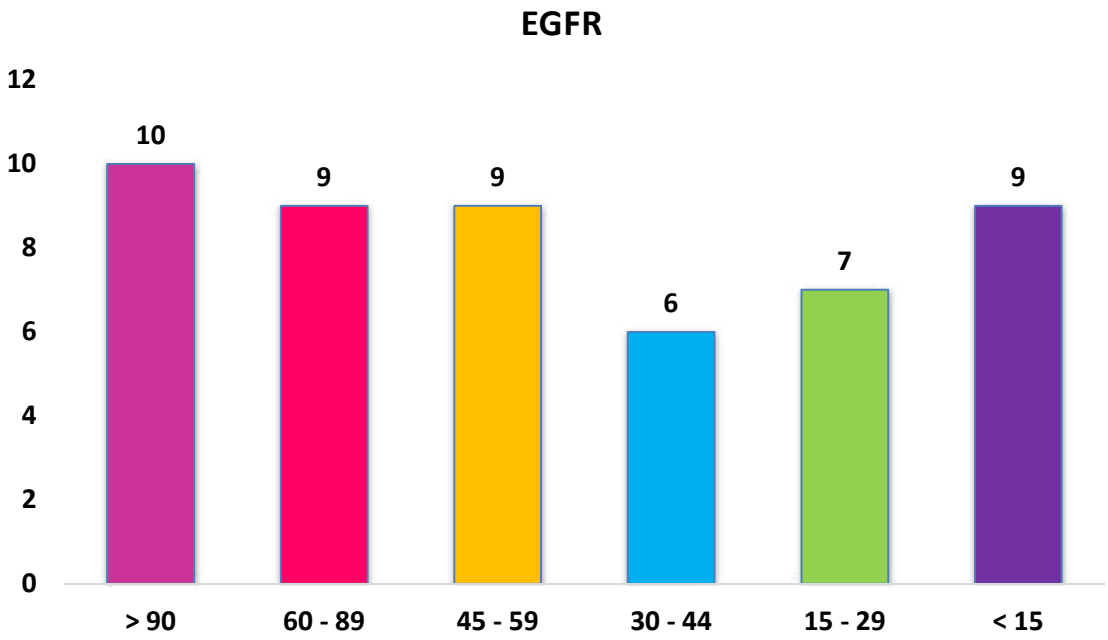
	SERUM CREATININE	PERCENTAGE
LESS THAN 1	14	28%
MORE THAN 1	36	72%



In our study serum creatinine was greater than 1 in 72% of individuals. Less than 1 in 28% of individuals.

ESTIMATED GLOMERULAR FILTRATION RATE

STAGE	EGFR	NO OF PATIENTS	PERCENTAGE
1	> 90	10	20%
2	60 – 89	9	18%
3a	45 – 59	9	18%
3b	30 – 44	6	12%
4	15 – 29	7	14%
5	< 15	9	18%



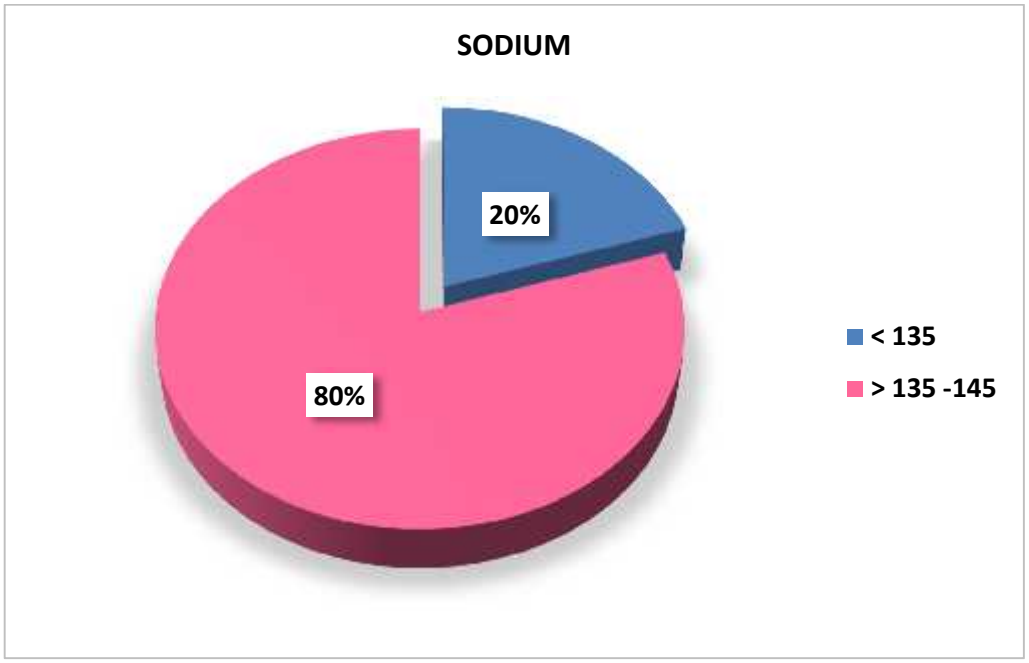
In our study EGFR was greater than 90 in 20% of individuals , 60 to 89 in 18% of individuals , 45 to 59 in 18% , 30 to 44 in 12% , 15 to 29 in 14% and less than 15 in 18% of individuals.

SERUM ELCTROLYTES

	SERUM ELECTROLYTES	
	MEAN	**STANDARD DEVIATION**
SODIUM	**137**	**4.57**
POTASSIUM	**4.52**	**0.58**

SODIUM

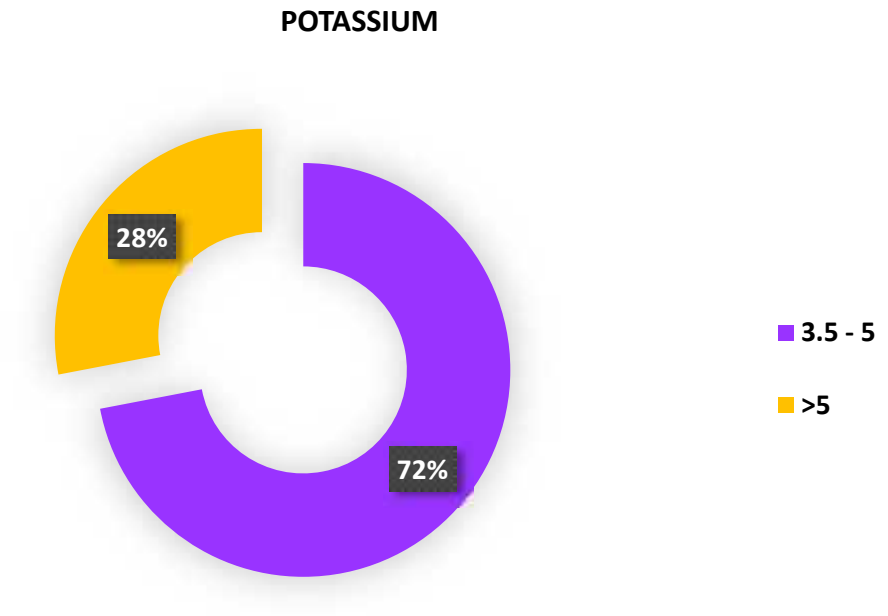
	SODIUM	PERCENTAGE
< 135	10	20%
> 135 -145	40	80%



In our study in 20% of individuals sodium levels were less than 135 mEq/L , between 135 to 145 mEq/L in 80% of individuals.

POTASSIUM

	POTASSIUM	PERCENTAGE
3.5 - 5	36	72%
>5	14	28%

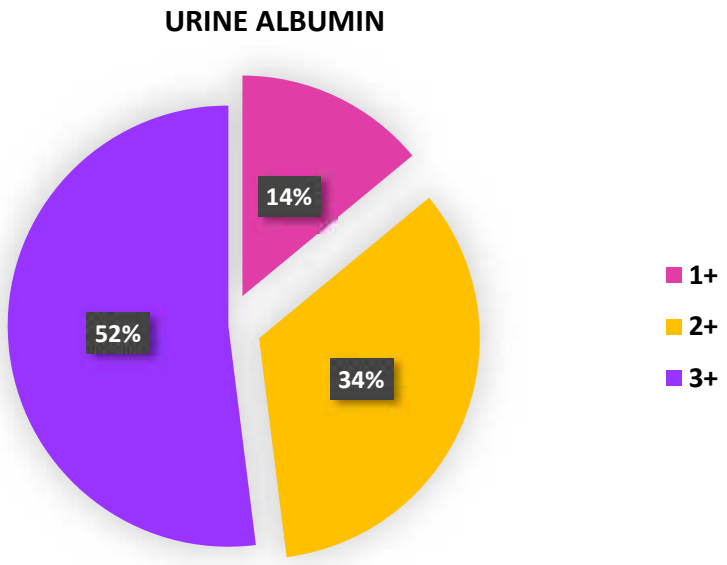


In our study 72% of individuals had serum potassium between 3.5 to 5mEq/L.

28% of individuals had serum potassium > 5mEq/L

URINE ALBUMIN

	URINE ALBUMIN	PERCENTAGE
1+	7	14%
2+	17	34%
3+	26	52%



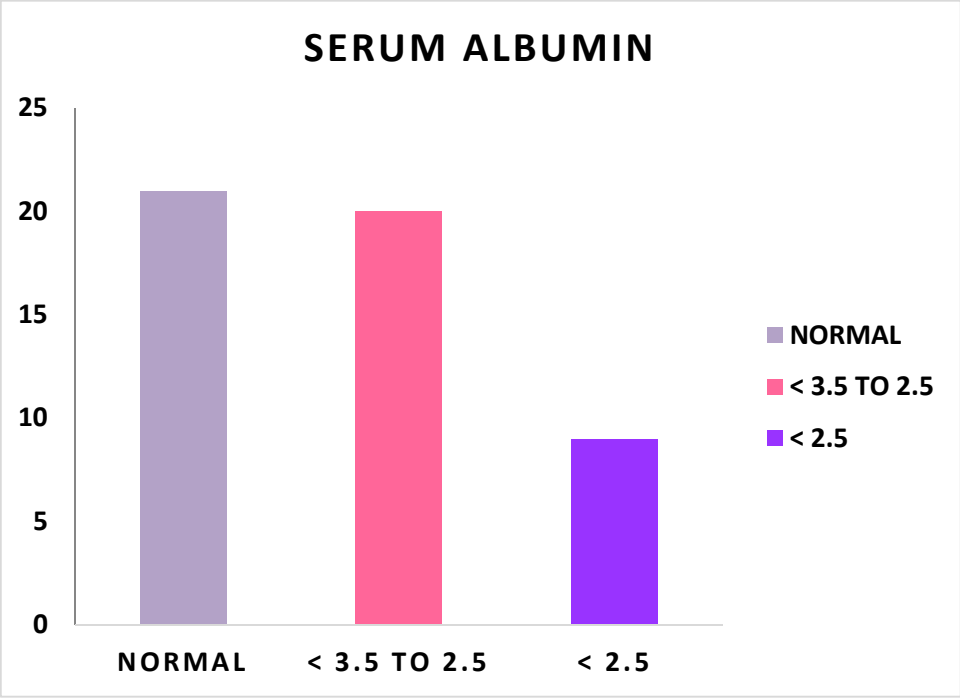
In our study urine albumin was 3+ in 52% of individuals , albumin was 2+ in 34% of individuals , 1+ in 14% of individuals.

LIVER FUNCTION TEST

	LIVER FUNCTION TEST	
	MEAN	**S.D**
BILIRUBIN - TOTAL	**1.11**	**0.48**
BILIRUBIN - DIRECT	**0.64**	**0.32**
BILIRUBIN - INDIRECT	**0.46**	**0.23**
SGOT	**34.9**	**13.67**
SGPT	**34.1**	**14.04**
ALP	**95.18**	**31.04**
TOTAL PROTEIN	**5.78**	**0.97**
ALBUMIN	**3.26**	**0.6**
GLOBULIN	**2.52**	**0.53**

SERUM ALBUMIN

	SERUM ALBUMIN	PERCENTAGE
NORMAL	21	42%
< 3.5 TO 2.5	20	40%
< 2.5	9	18%



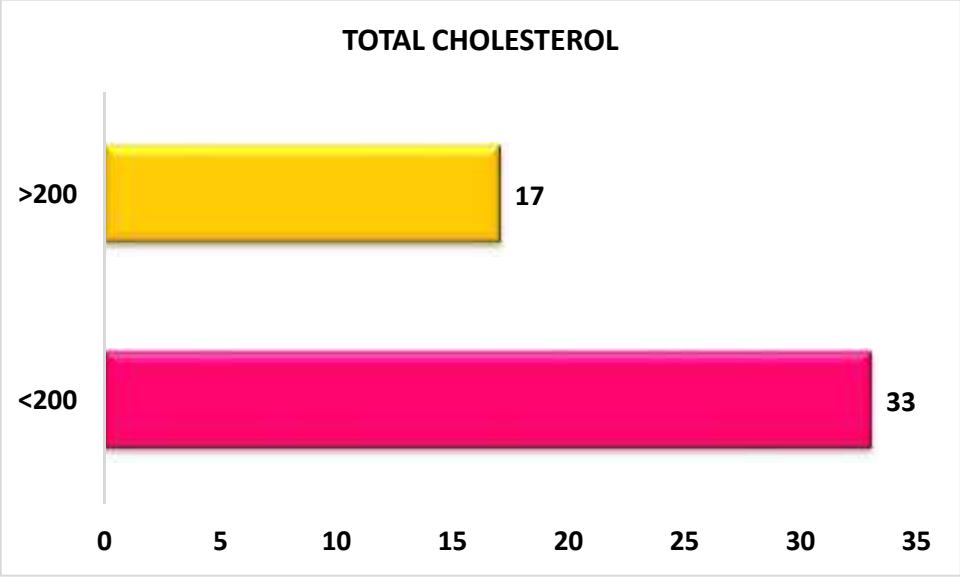
In our study serum albumin was normal in 42% of individuals , <3.5 to 2.5 g/dl in 40% of individuals , <2.5 g/dl in 18% of individuals.

LIPID PROFILE

	LIPID PROFILE	
	MEAN	**STANDARD DEVIATION**
TOTAL CHOLESTEROL	**211.88**	**118.44**
LDL	**128.68**	**92.43**
HDL	**47.02**	**7.11**
TRIGLYCERIDES	**149.74**	**43.69**
VLDL	**33.02**	**14.66**

TOTAL CHOLESTEROL

	TOTAL CHOLESTEROL	PERCENTAGE
<200	33	66%
>200	17	34%

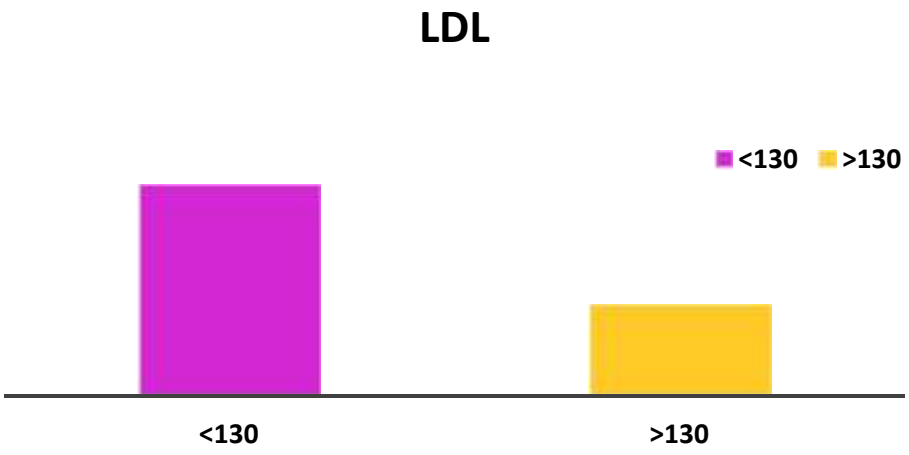


In our study total cholesterol was leass than 200mg/dL in 66% of individuals.

Greater than 200 mg/dl in 33% of individuals.

LDL

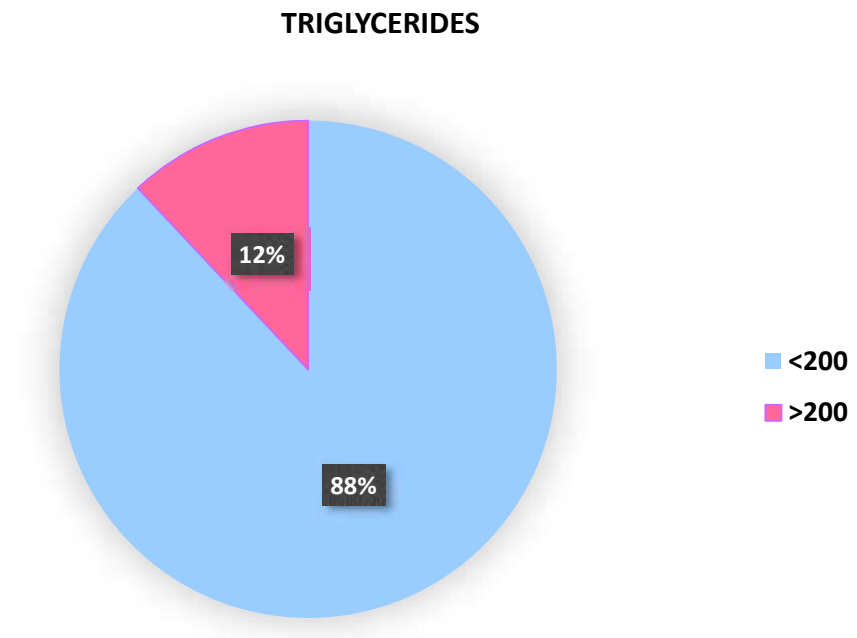
	LDL	PERCENTAGE
<130	**35**	**70%**
>130	**15**	**30%**



In our study LDL was less than 130mg/dl in 70% of individuals. More than 130mg/dl in 30% of individuals.

TRIGLYCERIDES

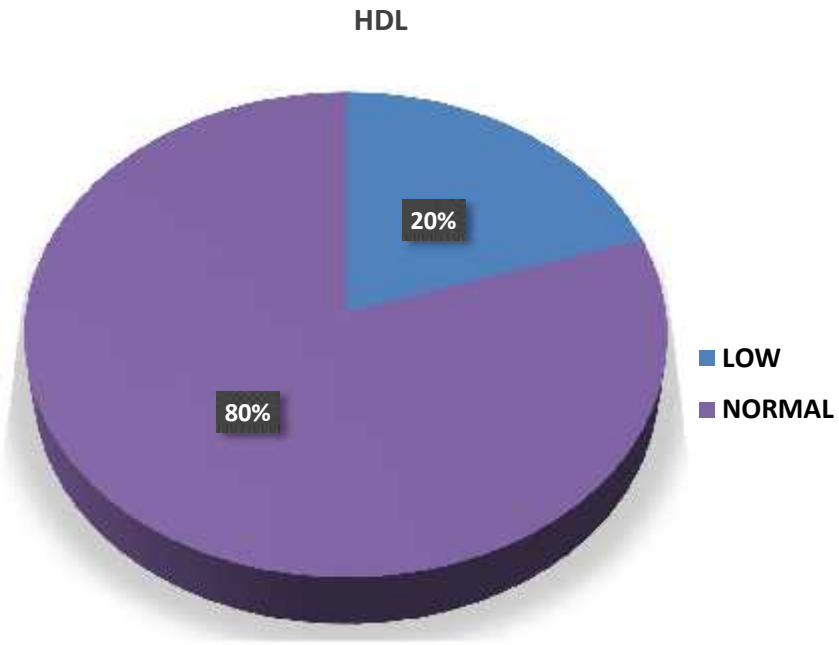
	TRIGLYCERIDES	PERCENTAGE
<200	44	88%
>200	6	12%



In our study triglycerides levels were <200 mg/dl in 88% of individuals, >200 mg/dl in 22% of individuals.

HDL

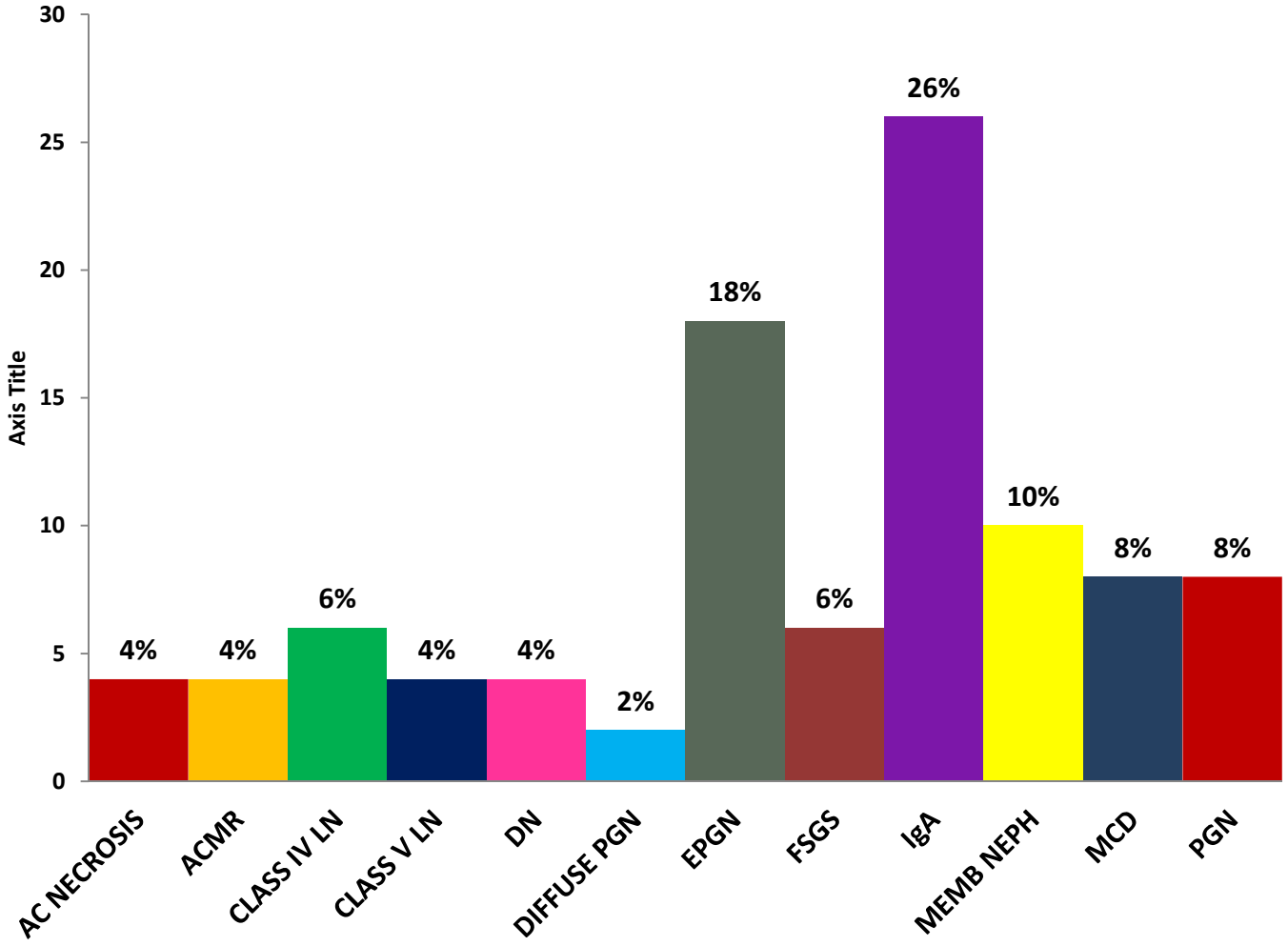
	HDL	PERCENTAGE
LOW	**10**	**20%**
NORMAL	**40**	**80%**



In our study HDL was normal in 80% of individuals. Low in 20% of individuals.

RENAL BIOPSY

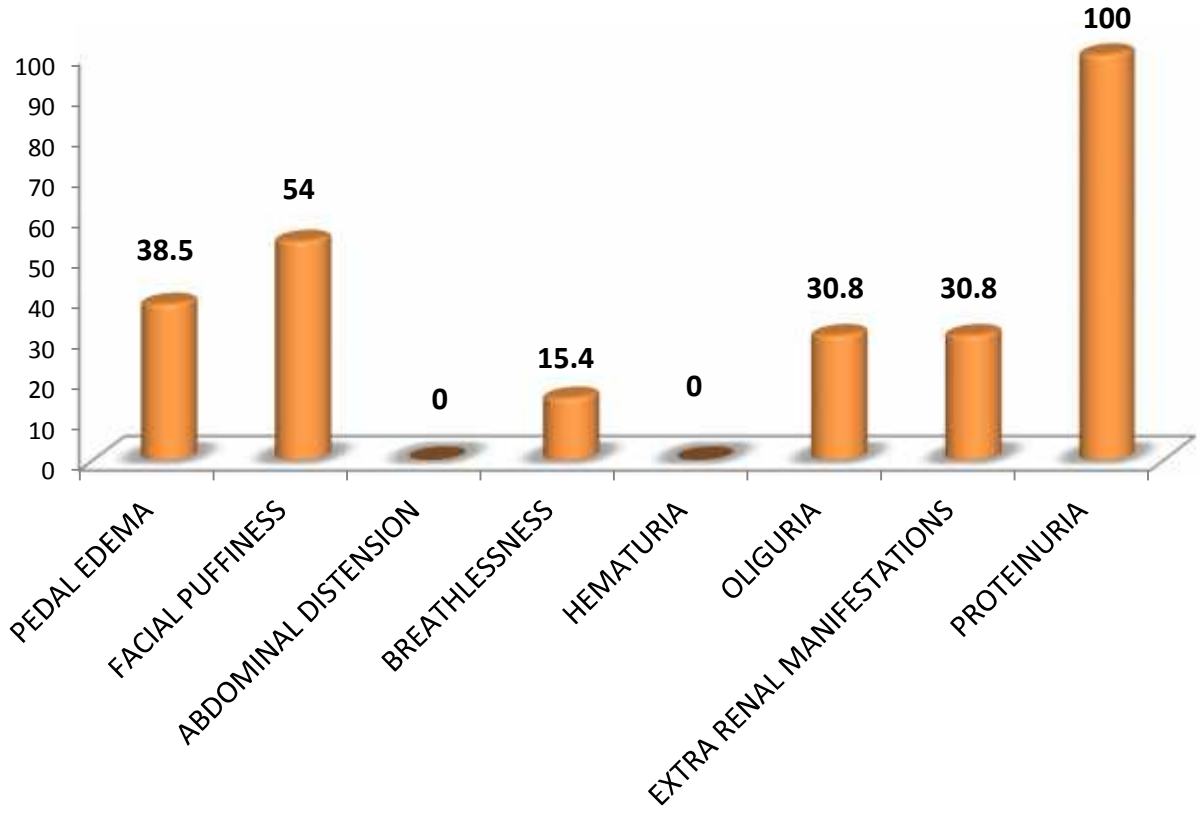
	NO OF PATIENTS	PERCENTAGE
ACN	2	4%
ACMR	2	4%
CLASS IV LN	3	6%
CLASS V LN	2	4%
DN	2	4%
DIFFUSE PGN	1	2%
EPGN	9	18%
FSGS	3	6%
IgA NEPHROPATHY	13	26%
MEMB NEPHROPATHY	5	10%
MCD	4	8%
PGN	4	8%



In our study IgA nephropathy was most common 26% , followed by Endocapillary proliferative glomerulonephritis which was about 18% .Next common was membranous nephropathy about 10% followed by minimal change disease and PGN which was 8% each.

IgA NEPHROPATHY : N=13

CLINICAL FEATURES	NO OF PATIENTS	PERCENTAGE
PEDAL EDEMA	5	38.5 %
FACIAL PUFFINESS	7	54 %
ABDOMINAL DISTENSION	0	0 %
BREATHLESSNESS	2	15.4 %
HEMATURIA	0	0 %
OLIGURIA	4	31 %
EXTRA RENAL MANIFESTATIONS	4	31 %
PROTEINURIA	13	100 %



In patients with IgA nephropathy everyone presented with proteinuria. 54% of them had facial puffiness and 38 % had pedal edema. Nearly 31% had oliguria and extra renal manifestations. None of them had hematuria.

DISCUSSION

The various forms of glomerulopathies have been discussed earlier. In this study , in 50 randomly selected patients renal biopsy has been done and the exact glomerular pathology has been ascertained. The etiological and clinical profile of the glomerular diseases has been evaluated.

Among the 50 patients 26 are males and 24 males are females. Most of the patients fall under 45 years of age , with maximum number under 30 years. This suggests the higher incidence of glomerular pathology in young age. Majority of patients have normal BMI. About half of the people have normal Blood pressure while the remaining half have raised BP (> 140/90 mmHg).

Nearly half have volume overload features like pedal edema and facial puffiness. But Breathlessness is seen in only 18% of patients. Hematuria is present in 4% of patients. Oliguria is noted in only 20 % of patients .

Extra renal manifestations include fever , joint pain , abdominal pain and headache . As a whole extra renal manifestations are present in only 34% of individuals. Among them most prevalent extra renal manifestation is joint pain and fever.

Nearly 10% have previous history of Diabetes mellitus and Hyertension. Only one patient is a known case of SLE.

78% are anaemic. This shows the correlation between renal pathology and anaemia. Blood urea is elevated (above 40) in 58% of individuals. Serum creatinine is more than 1 in 72 % of individuals.

eGFR is less than 15mL/min/1.73m^2 in 18% of patients. Hyponatremia is present in 20% and hyperkalemia is present in 28% of patients. Albuminuria is present in half of the patients which is pathognomonic of glomerular injury. Serum albumin is reduced in 58% of patients. On reviewing the lipid profile total cholesterol is raised in 34% , LDL raised in 30% and Triglycerides raised in 12% of patients. Serum HDL is normal in 80% of people.

Renal biopsy was done. The most common glomerular pathohlogy is IgA nephropathy seen in 13 patients (26%) . Among patients with IgA nephropathy volume overloaded states like pedal edema and facial puffiness is seen in most of the patients. Proteinuria is seen in all the patients. About 31% have oliguria. But none have hematuria. Extra renal manifestations is present in 31% of patients.

Second common pathology encountered is Endocapillary proliferative glomerulonephritis (EPGN) which is seen in 9 patients (18%) .EPGN is

a histopathological entity seen in lupus nephritis , post infective gomerulonephritis and MPGN. In our study EPGN is seen in 8 cases of post infective glomerulonephritis and 1 case of lupus nephritis.

The next common pathologies are membranous nephropathy and lupus nephritis each present in 10% of patients. It is followed by minimal change disease and proliferative glomerulonephritis (8% each). Focal segmental glomerulosclerosis is present in 6% of patients. Diabetic nephropathy , acute cortical necrosis and acute cell mediated rejection are each present in 4 % of patients. Finally Diffuse proliferative glomerulonephritis is seen in 2% of patients.

CONCLUSION

In this study among the 50 patients evaluated IgA nephropathy is the most common presentation noted. Proteinuria is present in all patients with IgA nephropathy. Volume overload state is present in nearly half of patients with IgA nephropathy. Second common presentation is post infective glomerulonephritis. Urine quantification of protein remains a simple yet reliable test for picking up clinically significant glomerular diseases.

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ANNEXURE : 1

ABBREVATIONS

BPRD : BIOPSY PROVEN RENAL DISEASE

PGD : PRIMARY GLOMERULAR DISEASE

TIN : TUBULOINTERSTITIAL NEPHRITIS

SGD : SECONDARY GLOMERULAR DISEASE

IMN : IDIOPATHIC MEMBRANOUS NEPHROPATHY

LN : LUPUS NEPHRITIS

MCD : MINIMAL CHANGE DISEASE

IGAN : IGA NEPHROPATHY

FSGS : FOCAL SEGMENTAL GLOMERULOSCLEROSIS

MGN : MEMBRANOUS GLEMERULOPATHY

EPGN : ENDOCAPILLARY PROLIFERATIVE GLOMERULONEPHRITIS

ACR : ACUTE CORTICAL NECROSIS

ACMR : ACUTE CELL MEDIATED REJECTION

DN : DIABETIC NEPHROPATHY

DPGN : DIFFUSE PROLIFERATIVE GLOMERULONEPHRITIS

PGN : PROLIFERATIVE GLOMERULONEPHRITIS

ANNEXURE : 2

PROFORMA

Name : Age : Sex :

Occupation :

Height : Weight : BMI :

PR – BP –

Clinical Presentation

Pedal Odema : Yes/ No

Facial Puffiness : Yes/ No

Abdominal Distension : Yes/ No

Breathlessness : Yes/ No

Haematuria : Yes/ No

Oliguria : Yes/ No

Extra Renal Manifestations : Yes/ No

Past History

DM____ SHT____ BA____ TB____ SLE____

RA____ Natural Medicine _____

Personal History

Diet – Mixed / Veg

Smoking –

Alcohol –

Investigation

CBC		Blood Urea	-		
TC	-	Serum Creatinine	-	Urine - Alb	
DC	-	Na^+	-	Sugar	
ESR	-	K^+	-	Deposits	
Hb	-				
Platlets	-				

LFT	**Lipid Profile**	HIV -
Total BR -	Total Cholesterol	HBSAG -
Direct -	LDL	Anti – HCV -
Indirect -	HDL	
SGOT -	TGS	**Others**
SGPT -	VLDL	
ALP -		
T.Protein -		
Albumin -		
Globulin -		

Renal Biopsy :

ANNEXURE 3 :MASTER CHART

S.NO	NAME	AGE	SEX	BMI	SBP	DBP	PEDAL EDEMA
1	NATARAJAN	49	M	21.3	140	90	ABSENT
2	MANIKARAJA	40	M	28.8	140	90	PRESENT
3	SIVAKUMAR	22	M	25.4	170	110	ABSENT
4	MOHAIDEEN	34	M	19.2	130	80	ABSENT
5	AYYAVU	60	M	23.2	150	100	PRESENT
6	VARGESE	71	M	23.2	150	100	PRESENT
7	RADHA	36	F	22.2	150	100	ABSENT
8	BHUVANESHWARAN	19	M	21.5	110	70	PRESENT
9	MUMTAZ BEGUM	40	F	23.4	140	80	ABSENT
10	RAJESWARI	39	F	25.8	130	80	ABSENT
11	SUBUTHAI	40	F	20	150	100	PRESENT
12	SIKIRTHA	31	F	24	160	90	PRESENT
13	MAHALINGAM	53	M	21.9	130	90	ABSENT
14	GANESAN	26	M	19.2	130	80	ABSENT
15	KOVILPITCHAI	62	M	21.9	140	90	ABSENT
16	CHELLAMMAL	47	F	22.7	140	90	PRESENT
17	FATHIISHA	16	F	16	120	80	PRESENT
18	ARUN	36	M	21.3	150	100	PRESENT
19	MUTHUSAMY	56	M	19.5	130	80	ABSENT
20	CHINNIAMMAL	55	F	20	130	80	ABSENT
21	BHAVANI	22	F	21.5	120	80	ABSENT
22	SHIFANA	19	F	20	110	70	ABSENT
23	AISHWARYA	25	F	20	120	80	PRESENT
24	RULINI	29	F	20	150	100	PRESENT
25	VALLINAYAGAM	65	M	23.5	200	110	ABSENT
26	SUDALAIKANI	55	M	23.5	180	100	ABSENT
27	RANGAMATHAN	45	M	22.4	180	130	ABSENT
28	RESHMA	40	F	22.9	150	90	PRESENT

29	RAMKI	17	M	17.8	100	60	PRESENT
30	MURUGESHWARI	30	F	22.7	130	90	PRESENT
31	PARVEEN	24	F	22.2	110	70	PRESENT
32	PRAVEENA MARY	22	F	22	100	60	ABSENT
33	AMUTHA	38	F	21.3	120	80	PRESENT
34	ANANDHAVALLI	40	F	21.3	120	80	PRESENT
35	MUTHULINGAM	55	M	16.2	130	90	PRESENT
36	VARSHINI	28	F	22.7	130	90	PRESENT
37	IMMANUEL	47	M	23.2	130	80	ABSENT
38	NATARAJAN	49	M	20.1	190	100	ABSENT
39	NALINI	25	F	21.5	140	80	ABSENT
40	MAHAMED AYUB KHAN	27	M	23.5	150	90	ABSENT
41	SAMUEL	45	M	23.2	150	90	ABSENT
42	LOGANATHAN	18	M	19.6	110	70	PRESENT
43	MESHACH	28	M	19.6	130	90	PRESENT
44	SUBBULAKSHMI	35	F	18.7	140	90	PRESENT
45	MUTHUMARI	27	F	25.3	120	80	PRESENT
46	MEENAKSHI	38	F	22.7	150	100	PRESENT
47	RAMAKRISHNAN	50	M	25	130	90	PRESENT
48	GOVIND	35	M	23	140	90	ABSENT
49	GANESAN	40	M	25.8	140	90	ABSENT
50	VANITHA	40	F	21.3	120	80	PRESENT

S.NO	NAME	FACIAL PUFFINESS	ABD DISTENSION	BREATHLESSNESS	HAEMATURIA	OLIGURIA	DM	SHT	BA
1	NATARAJAN	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
2	MANIKARAJA	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
3	SIVAKUMAR	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
4	MOHAIDEEN	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
5	AYYAVU	PRESENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	NO	NO
6	VARGESE	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	YES	NO	NO
7	RADHA	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	NO
8	BHUVANESHWARAN	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
9	MUMTAZ BEGUM	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	NO
10	RAJESWARI	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
11	SUBUTHAI	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
12	SIKIRTHA	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
13	MAHALINGAM	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	NO	NO
14	GANESAN	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
15	KOVILPITCHAI	ABSENT	PRESENT	ABSENT	ABSENT	PRESENT	NO	NO	NO
16	CHELLAMMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
17	FATHIISHA	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
18	ARUN	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	NO
19	MUTHUSAMY	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	NO	NO
20	CHINNIAMMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	NO	NO
21	BHAVANI	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
22	SHIFANA	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
23	AISHWARYA	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
24	RULINI	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
25	VALLINAYAGAM	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	YES	YES	NO

26	SUDALAIKANI	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	YES	YES	NO
27	RANGAMATHAN	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	NO	NO
28	RESHMA	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
29	RAMKI	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
30	MURUGESHWARI	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
31	PARVEEN	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
32	PRAVEENA MARY	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
33	AMUTHA	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	NO
34	ANANDHAVALLI	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	NO
35	MUTHULINGAM	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
36	VARSHINI	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	NO
37	IMMANUEL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	NO	NO
38	NATARAJAN	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
39	NALINI	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
40	MAHAMED AYUB KHAN	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
41	SAMUEL	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	YES	NO	NO
42	LOGANATHAN	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
43	MESHACH	PRESENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	NO	NO
44	SUBBULAKSHMI	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
45	MUTHUMARI	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	NO
46	MEENAKSHI	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	NO	NO	NO
47	RAMAKRISHNAN	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	YES	NO
48	GOVIND	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	NO
49	GANESAN	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	NO
50	VANITHA	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	NO

S.NO	NAME	EXTRA RENAL MANIFESTATION	EXTRA RENAL MANIFESTATION	TB	SLE	RA	NATURAL MEDICINE	SMOKING
1	NATARAJAN	PRESENT	GRADE 1 CKD/PALPITATION	NO	NO	NO	NO	NO
2	MANIKARAJA	ABSENT		NO	NO	NO	NO	NO
3	SIVAKUMAR	PRESENT	BELLS PALSY LT SIDE	NO	NO	NO	NO	NO
4	MOHAIDEEN	PRESENT	LOSS OF APPETITE	NO	NO	NO	NO	YES
5	AYYAVU	ABSENT		NO	NO	NO	NO	NO
6	VARGESE	ABSENT		NO	NO	NO	NO	NO
7	RADHA	ABSENT		NO	NO	NO	NO	NO
8	BHUVANESHWARAN	ABSENT		NO	NO	NO	NO	NO
9	MUMTAZ BEGUM	ABSENT		NO	NO	NO	NO	NO
10	RAJESWARI	PRESENT	BURNING MICTURATION	NO	NO	NO	NO	NO
11	SUBUTHAI	ABSENT		NO	NO	NO	NO	NO
12	SIKIRTHA	ABSENT		NO	NO	NO	NO	NO
13	MAHALINGAM	ABSENT		NO	NO	NO	NO	NO
14	GANESAN	PRESENT	NUMBNESS OF BOTH HANDS	NO	NO	NO	YES	NO
15	KOVILPITCHAI	ABSENT		NO	NO	NO	NO	NO
16	CHELLAMMAL	ABSENT		NO	NO	NO	NO	NO
17	FATHIISHA	ABSENT		NO	NO	NO	NO	NO
18	ARUN	ABSENT		NO	NO	NO	NO	NO
19	MUTHUSAMY	PRESENT	H/O SNAKE BITE,B/L PTOSIS	NO	NO	NO	NO	NO
20	CHINNIAMMAL	PRESENT	H/O SNAKE BITE,B/L PTOSIS	NO	NO	NO	NO	NO
21	BHAVANI	PRESENT	JOINT STIFFNESS/CHEEK PIGMENTATION	NO	NO	NO	NO	NO

22	SHIFANA	PRESENT	FEVER/JOINT STIFFNESS	NO	NO	NO	NO	NO
23	AISHWARYA	ABSENT		NO	NO	NO	NO	NO
24	RULINI	ABSENT		NO	NO	NO	NO	NO
25	VALLINAYAGAM	ABSENT		NO	NO	NO	NO	NO
26	SUDALAIKANI	ABSENT		NO	NO	NO	NO	NO
27	RANGAMATHAN	ABSENT		NO	NO	NO	NO	NO
28	RESHMA	ABSENT		NO	NO	NO	NO	NO
29	RAMKI	ABSENT		NO	NO	NO	NO	NO
30	MURUGESHWARI	ABSENT		NO	NO	NO	NO	NO
31	PARVEEN	ABSENT		NO	NO	NO	NO	NO
32	PRAVEENA MARY	PRESENT	SUPPURATIVE LESION	NO	YES	NO	NO	NO
33	AMUTHA	ABSENT		NO	NO	NO	NO	NO
34	ANANDHAVALLI	ABSENT		NO	NO	NO	NO	NO
35	MUTHULINGAM	PRESENT		NO	NO	NO	NO	NO
36	VARSHINI	ABSENT		NO	NO	NO	NO	NO
37	IMMANUEL	PRESENT	WEIGHT LOSS	NO	NO	NO	NO	NO
38	NATARAJAN	PRESENT	VOMITTING	NO	NO	NO	NO	NO
39	NALINI	PRESENT	FEVER/JOINT STIFFNESS	NO	NO	NO	NO	NO
40	MAHAMED AYUB KHAN	PRESENT	ABDOMINAL PAIN	NO	NO	NO	NO	NO
41	SAMUEL	PRESENT	FEVER/WEIGHT LOSS	NO	NO	NO	NO	NO
42	LOGANATHAN	ABSENT		NO	NO	NO	NO	NO
43	MESHACH	ABSENT		NO	NO	NO	NO	NO
44	SUBBULAKSHMI	PRESENT		NO	NO	NO	NO	NO
45	MUTHUMARI	ABSENT		NO	NO	NO	NO	NO
46	MEENAKSHI	ABSENT		NO	NO	NO	NO	NO

47	**RAMAKRISHNAN**	**ABSENT**		**NO**	**NO**	**NO**	**NO**	**NO**
48	**GOVIND**	**ABSENT**		**NO**	**NO**	**NO**	**NO**	**NO**
49	**GANESAN**	**ABSENT**		**NO**	**NO**	**NO**	**NO**	**NO**
50	**VANITHA**	**ABSENT**		**NO**	**NO**	**NO**	**NO**	**NO**

S.NO	NAME	ALCOHOL	RBS	TOTAL COUNT	NEUTROPHIL	EOSINOPHIL	LEUKOCYTE	ESR/HR	HB	PCV	PLATELET
1	NATARAJAN	NO	138	8100	60	5	35	7	9.3	27.6	310000
2	MANIKARAJA	NO	141	6800	61	6	33	40	9		89000
3	SIVAKUMAR	NO	128	19400	40	20	40	25	9.6		65000
4	MOHAIDEEN	YES	151	13600	96	0	4	70	5.3		419000
5	AYYAVU	NO	150	12000	75	5	20	58	13.6		450000
6	VARGESE	YES	250	10900	75	5	20	46	13.7		450000
7	RADHA	NO	120	13900	85	0	15	20	9.1		223000
8	BHUVANESHWARAN	NO	70	6700	47	1	42	50	14.3	42.9	396000
9	MUMTAZ BEGUM	NO	141	14000	89	5	6	74	8.8	27	250000
10	RAJESWARI	NO	105	8100	89	5	6	15	8.7	26.8	260000
11	SUBUTHAI	NO	146	5600	67	9	24	68	9.5		190000
12	SIKIRTHA	NO	142	4500	42	0	58	120	7		423000
13	MAHALINGAM	NO	75	11800	73	4	23	90	9.8	27.5	429000
14	GANESAN	NO	114	6800	44	20	36	100	10.2	48.2	334000
15	KOVILPITCHAI	NO	150	6600	63	3	24	78	9.7		204000
16	CHELLAMMAL	NO	135	2300	52	7	41	130	10.2	32	527000
17	FATHIISHA	NO	140	14000	82	2	16	32	10.1		170000
18	ARUN	NO	135	14000	86	0	14	24	9.2		250000
19	MUTHUSAMY	NO	235	23000	89	5	6	56	12		80000
20	CHINNIAMMAL	NO	240	24000	87	5	8	44	11.6		85000
21	BHAVANI	NO	130	7500	60	6	34	150	10.2	30.1	350000
22	SHIFANA	NO	142	7500	60	6	36	140	10.6		300000
23	AISHWARYA	NO	94	8000	45	10	45	15	13.8	38	400000
24	RULINI	NO	107	13900	85	0	15	20	9.1		223000
25	VALLINAYAGAM	NO	172	7500	65	2	33	45	11.5	32.5	280000
26	SUDALAIKANI	NO	139	8000	65	3	32	42	11.5	33.5	282000
27	RANGAMATHAN	NO	121	14300	71	12	17	40	18.1		326000

28	RESHMA	NO	120	14300	71	12	17	40	13.2		350000
29	RAMKI	NO	115	12200	64	6	30	30	10.8		371000
30	MURUGESHWARI	NO	105	2400	65	13	22	90	7.4		114000
31	PARVEEN	NO	121	8500	84	4	12	68	8.2		150000
32	PRAVEENA MARY	NO	112	7600	83	4	13	110	6.4	17.8	140000
33	AMUTHA	NO	141	12000	80	0	20	45	8.9		350000
34	ANANDHAVALLI	NO	112	11900	81	0	19	40	6.3		434000
35	MUTHULINGAM	NO	120	7500	67	9	24	38	9.2		200000
36	VARSHINI	NO	111	5700	66	0	34	54	7.1		127000
37	IMMANUEL	NO	120	12500	74	0	26	60	7.5		450000
38	NATARAJAN	NO	152	8100	60	5	35	70	9.3		310000
39	NALINI	NO	115	6500	86	3	11	80	8		117000
40	MAHAMED AYUB KHAN	NO	115	16100	70	4	26	40	10.6		170000
41	SAMUEL	NO	189	9000	74	0	26	45	7.5		264000
42	LOGANATHAN	NO	80	6800	48	10	42	40	13.6	43	350000
43	MESHACH	NO	121	8600	43	11	46	135	10.2		370000
44	SUBBULAKSHMI	NO	115	21000	75	9	14	40	9.5		250000
45	MUTHUMARI	NO	125	6200	43	11	46	135	10.6		374000
46	MEENAKSHI	NO	141	11000	60	5	35	70	9.4		350000
47	RAMAKRISHNAN	NO	115	12000	73	4	23	45	9.9		450000
48	GOVIND	NO	130	13500	85	0	15	23	9		250000
49	GANESAN	NO	121	14000	85	0	15	36	10		270000
50	VANITHA	NO	112	11900	81	0	19	40	6.3		434000

S.NO	NAME	UREA	CREATININE	EGFR	SODIUM	POTASSIUM	URINE ALB	URINE RBC	URINE SUG	TOTAL BR	DIRECT
1	NATARAJAN	37	3.1	22	136	5.1	2+	NIL	TRACE	1.7	0.9
2	MANIKARAJA	44	1.7	45	141	6	3+	NIL	TRACE	1.1	0.6
3	SIVAKUMAR	35	2.5	32	145	3.7	1+	4 TO 6	NIL	0.7	0.4
4	MOHAIDEEN	128	2.9	22	142	4.2	2+	NIL	NIL	1.4	0.8
5	AYYAVU	34	1.2	62	136	4.8	3+	NIL	NIL	0.7	0.4
6	VARGESE	35	1	74	125	3.5	3+	NIL	NIL	0.7	0.4
7	RADHA	107	4.9	10	137	4.2	3+	1TO 2	NIL	0.4	0.2
8	BHUVANESHWARAN	17	0.6	174	143	4.9	2+	NIL	NIL	0.6	0.3
9	MUMTAZ BEGUM	40	1.7	33	132	3.9	1+	NIL	NIL	1.3	0.8
10	RAJESWARI	40	1.7	33	132	3.9	1+	4 TO 5	NIL	2.1	1.6
11	SUBUTHAI	45	1.3	45	135	4.1	1+	8 TO 10	NIL	1.4	0.8
12	SIKIRTHA	50	1.1	58	140	5.6	3+	NIL	NIL	0.6	0.3
13	MAHALINGAM	51	1.5	49	120	4	2+	NIL	NIL	1.2	0.6
14	GANESAN	16	0.7	136	137	4.2	3+	NIL	NIL	1.2	0.6
15	KOVILPITCHAI	150	2.6	25	136	5.4	2+	NIL	NIL	0.9	0.6
16	CHELLAMMAL	21	0.6	107	133	4.3	2+	NIL	NIL	1.8	1.1
17	FATHIISHA	49	1.6	43	136	5.1	2+	NIL	NIL	1.2	0.8
18	ARUN	120	5	13	137	4.2	3+	NIL	NIL	0.5	0.2
19	MUTHUSAMY	80	6.9	6	132	4.2	1+	6 TO 10	3+	0.6	0.3
20	CHINNIAMMAL	82	7.9	5	132	4.5	2+	NIL	NIL	0.6	0.3
21	BHAVANI	20	0.8	90	143	4.2	2+	NIL	NIL	1.6	0.9
22	SHIFANA	18	0.6	129	145	4.5	2+	NIL	NIL	1.4	0.8
23	AISHWARYA	41	1.2	55	139	4.2	3+	NIL	NIL	0.4	0.2
24	RULINI	107	4.9	10	137	4.2	3+	NIL	NIL	0.4	0.2
25	VALLINAYAGAM	54	2.1	32	139	5.1	3+	NIL	2+	1.3	0.8

26	SUDALAIKANI	47	2.7	25	136	4.7	3+	NIL	NIL	1.2	0.6
27	RANGAMATHAN	19	1.1	72	136	5.1	3+	NIL	NIL	1.7	0.6
28	RESHMA	42	0.9	69	141	4.2	2+	NIL	NIL	1.7	0.6
29	RAMKI	41	0.7	149	140	4.7	3+	NIL	NIL	1.7	0.7
30	MURUGESHWARI	66	1.6	38	139	5.5	2+	NIL	NIL	1.3	0.8
31	PARVEEN	17	0.8	88	133	5.4	2+	NIL	NIL	1	0.8
32	PRAVEENA MARY	17	0.8	90	133	5.5	2+	NIL	NIL	1.7	1
33	AMUTHA	80	4.2	12	136	4.2	3+	NIL	NIL	1.6	1
34	ANANDHAVALLI	57	3.4	15	136	4.5	3+	NIL	NIL	1.8	1.2
35	MUTHULINGAM	42	1.2	63	136	3.7	1+	NIL	NIL	1.3	0.7
36	VARSHINI	101	11	49	141	4.4	3+	NIL	NIL	1.7	1
37	IMMANUEL	18	0.8	73	142	4	3+	NIL	NIL	1.3	0.8
38	NATARAJAN	37	3.1	104	136	5.1	2+	NIL	NIL	0.4	0.2
39	NALINI	53	1.1	61	133	3.9	3+	NIL	NIL	1.4	1
40	MAHAMED AYUB KHAN	32	1.5	56	136	4.1	3+	NIL	NIL	1.2	0.7
41	SAMUEL	74	2.5	21	142	4	3+	NIL	NIL	1.3	0.8
42	LOGANATHAN	17	0.8	126	144	4.7	3+	NIL	NIL	0.6	0.3
43	MESHACH	24	0.8	115	139	5.2	3+	NIL	NIL	0.8	0.6
44	SUBBULAKSHMI	47	1.2	51	138	4.3	1+	NIL	NIL	1.2	0.8
45	MUTHUMARI	24	0.8	86	139	5.2	3+	NIL	NIL	0.8	0.6
46	MEENAKSHI	37	3.1	23	136	5.1	2+	NIL	NIL	0.4	0.2
47	RAMAKRISHNAN	180	4.5	14	138	3.7	2+	NIL	NIL	1.1	0.7
48	GOVIND	130	5.6	12	137	4.3	3+	NIL	NIL	0.4	0.2

49	**GANESAN**	**130**	**4.8**	**14**	**137**	**4.2**	**3+**	**NIL**	**NIL**	**0.4**	**0.2**
50	**VANITHA**	**57**	**3.4**	**49**	**136**	**4.5**	**3+**	**NIL**	**NIL**	**1.8**	**1.2**

S.NO	NAME	INDIRECT	SGOT	SGPT	ALP	T.PROTEIN	ALBUMIN	GLOBULIN	TC	LDL	HDL	TGL
1	NATARAJAN	0.8	40	42	104	6.8	3.6	3.2	251	162	54	172
2	MANIKARAJA	0.5	38	42	104	6.2	3.2	3	110	30	30	150
3	SIVAKUMAR	0.3	35	37	76	5.1	3.1	2	145	106	52	250
4	MOHAIDEEN	0.6	52	48	98	6.6	3.6	3	112	59	45	150
5	AYYAVU	0.3	20	27	86	4	2	2	590	500	50	200
6	VARGESE	0.3	20	27	86	4	2	2	490	500	50	200
7	RADHA	0.2	16	15	49	5.5	3.5	2	112	59	45	150
8	BHUVANESHWARAN	0.3	26	29	84	4.4	2.4	2	395	219	47	210
9	MUMTAZ BEGUM	0.5	38	42	104	5.6	3	2.6	121	48	45	150
10	RAJESWARI	0.5	54	52	106	5.6	3	2.6	112	58	44	145
11	SUBUTHAI	0.6	36	34	104	5.3	3.3	2	135	74	40	108
12	SIKIRTHA	0.3	14	18	81	5.9	3.5	2.4	111	57	44	151
13	MAHALINGAM	0.6	34	32	91	4.4	2.4	2	111	57	44	151
14	GANESAN	0.6	21	32	94	4	2	2	360	260	50	250
15	KOVILPITCHAI	0.3	42	44	104	7.2	4.2	3	219	162	52	164
16	CHELLAMMAL	0.7	42	41	107	5	2.5	2.5	300	118	75	201
17	FATHIISHA	0.4	42	40	110	6.6	3.6	3	640	220	54	288
18	ARUN	0.3	16	15	49	5.5	3.5	2	112	56	43	150
19	MUTHUSAMY	0.3	39	15	145	5	3	2	114	55	42	149
20	CHINNIAMMAL	0.3	39	14	145	5	3	2	160	112	38	89
21	BHAVANI	0.7	61	62	104	6.8	3.4	3.4	210	168	45	120
22	SHIFANA	0.6	52	47	102	6.8	3.4	3.4	178	116	35	120
23	AISHWARYA	0.2	24	20	80	4.4	2.4	2	350	210	54	188
24	RULINI	0.2	16	15	49	5.5	3.5	2	202	142	40	168
25	VALLINAYAGAM	0.5	25	24	94	5.7	3.1	2.6	154	108	52	126
26	SUDALAIKANI	0.6	26	22	106	5	3	2	168	112	52	120
27	RANGAMATHAN	1.1	41	44	93	6.8	4.4	2.4	154	108	52	126

28	RESHMA	1.1	41	44	93	6.8	4.4	2.4	172	120	56	118
29	RAMKI	1	26	32	248	8	4	4	200	40	40	200
30	MURUGESHWARI	0.5	40	42	104	5	3	2	170	120	52	120
31	PARVEEN	0.2	32	32	106	6.8	3.8	3	110	78	52	110
32	PRAVEENA MARY	0.7	73	37	102	6.3	2.9	3.4	150	116	52	110
33	AMUTHA	0.6	38	40	102	7	4	3	169	112	52	120
34	ANANDHAVALLI	0.6	37	64	102	6.8	3.8	3	180	110	42	123
35	MUTHULINGAM	0.6	63	47	102	5.3	3.3	2	175	74	40	108
36	VARSHINI	0.7	52	51	68	6.4	3.4	3	161	112	48	130
37	IMMANUEL	0.5	41	43	116	6.2	3.8	2.4	247	134	51	110
38	NATARAJAN	0.2	18	10	109	6	3	3	251	153	47	172
39	NALINI	0.4	43	44	69	7	4.2	2.8	152	106	50	110
40	MAHAMED AYUB KHAN	0.5	17	22	72	6.8	3.7	3.1	160	106	46	108
41	SAMUEL	0.5	44	42	110	7	4	3	210	116	44	108
42	LOGANATHAN	0.3	26	19	84	4.4	2.4	2	405	223	56	210
43	MESHACH	0.2	40	38	94	5.7	3.1	2.6	250	160	52	163
44	SUBBULAKSHMI	0.4	38	36	61	5.3	3.3	2	168	74	40	108
45	MUTHUMARI	0.2	40	38	94	5.7	3.1	2.6	220	160	52	180
46	MEENAKSHI	0.2	18	10	109	6	3	3	275	120	42	180
47	RAMAKRISHNAN	0.4	40	40	57	4.4	2.4	2	168	74	40	108
48	GOVIND	0.2	16	15	50	5.5	3.5	2	164	75	41	110
49	GANESAN	0.2	16	15	50	5.5	3.5	2	141	65	40	112
50	VANITHA	0.6	37	64	102	6.8	3.8	3	180	110	42	123

S.NO	NAME	VLDL	HIV	HBSAG	ANTI HCV	USG	RENAL BIOPSY
1	NATARAJAN	32	NEG	NEG	NEG		IgA Nephropathy
2	MANIKARAJA	50	NEG	NEG	NEG		IgA Nephropathy
3	SIVAKUMAR	32	NEG	NEG	NEG	ENLARGED KIDNEY WITH CORTICAL ECHOES	LYMPHOPRLIFERATIVE DISEASE/IgA Nephropathy
4	MOHAIDEEN	25	NEG	NEG	NEG		ENDOCAPILLARY PROLIFERATIVE GLOMERYLONEPHRITIS ASS WITH INFECTION
5	AYYAVU	40	NEG	NEG	NEG		MEMBRANOUS NEPHROPATHY
6	VARGESE	40	NEG	NEG	NEG	VESICAL CALCULUS	MEMBRANOUS NEPHROPATHY
7	RADHA	25	NEG	NEG	NEG		IgA Nephropathy
8	BHUVANESHWARAN	56	NEG	NEG	NEG	LT KIDNEY-MINIMAL CORTICAL CHANGE	MINIMAL CHANGE DISEASE
9	MUMTAZ BEGUM	26	NEG	NEG	NEG		ALLOGRAFT BIOPSY-ACUTE CELL MEDIATED REJECTION-BANFF 1A
10	RAJESWARI	27	NEG	NEG	NEG		ALLOGRAFT BIOPSY-ACUTE CELL MEDIATED REJECTION-BANFF 1A
11	SUBUTHAI	21	NEG	NEG	NEG	B/L PLEURAL EFFUSION	ENDOCAPILLARY PROLIFERATIVE GLOMERYLONEPHRITIS ASS WITH INFECTION
12	SIKIRTHA	21	NEG	NEG	NEG		ENDOCAPILLARY PROLIFERATIVE GLOMERYLONEPHRITIS ASS WITH INFECTION
13	MAHALINGAM	21	NEG	NEG	NEG	BOTH KIDNEY INCREASED ECHOGENICITY	ENDOCAPILLARY PROLIFERATIVE GLOMERYLONEPHRITIS ASS WITH INFECTION
14	GANESAN	50	NEG	NEG	NEG		MEMBRANOUS NEPHROPATHY
15	KOVILPITCHAI	32	NEG	NEG	NEG	B/L GRADE 1 CKD	ENDOCAPILLARY PROLIFERATIVE GLOMERYLONEPHRITIS ASS WITH INFECTION

16	CHELLAMMAL	32	NEG	NEG	NEG		MINIMAL CHANGE DISEASE
17	FATHIISHA	32	NEG	NEG	NEG		FSGS TIP VARIANCE
18	ARUN	22	NEG	NEG	NEG		IgA Nephropathy
19	MUTHUSAMY	23	NEG	NEG	NEG		ACUTE CORTICAL NECROSIS
20	CHINNIAMMAL	36	NEG	NEG	NEG		ACUTE CORTICAL NECROSIS
21	BHAVANI	40	NEG	NEG	NEG		CLASS V LUPUS NEPHRITIS/MEMBRANOUS NEPHROPATHY
22	SHIFANA	32	NEG	NEG	NEG		CLASS V LUPUS NEPHRITIS/MEMBRANOUS NEPHROPATHY
23	AISHWARYA	33	NEG	NEG	NEG	RT KIDNEY-MINIMAL CORTICAL CHANGE	MINIMAL CHANGE DISEASE
24	RULINI	32	NEG	NEG	NEG		IgA Nephropathy
25	VALLINAYAGAM	32	NEG	NEG	NEG	RT PLEURAL EFFUSION/MRD	FSGS /MODERATE ARTERISCLEROSIS
26	SUDALAIKANI	32	NEG	NEG	NEG		FSGS /MODERATE ARTERISCLEROSIS
27	RANGAMATHAN	32	NEG	NEG	NEG		IgA Nephropathy
28	RESHMA	32	NEG	NEG	NEG		IgA Nephropathy
29	RAMKI	120	NEG	NEG	NEG		ENDOCAPILLARY PROLIFERATIVE GLOMERYLONEPHRITIS ASS WITH INFECTION
30	MURUGESHWARI	32	NEG	NEG	NEG		DIFFUSE PROLIFERATIVE LUPUS NEPHRITIS
31	PARVEEN	32	NEG	NEG	NEG		LUPUS NEPHRITIS CLASS IV
32	PRAVEENA MARY	29	NEG	NEG	NEG		LUPUS NEPHRITIS CLASS IV
33	AMUTHA	32	NEG	NEG	NEG		PROGRESSING GLOMERULONEPHRITIS/SCLEROSING GLOMERULONEPHRITIS
34	ANANDHAVALLI	28	NEG	NEG	NEG		PROGRESSING GLOMERULONEPHRITIS/SCLEROSING GLOMERULONEPHRITIS

35	MUTHULINGAM	21	NEG	NEG	NEG		ENDOCAPILLARY PROLIFERATIVE GLOMERYLONEPHRITIS ASS WITH INFECTION
36	VARSHINI	32	NEG	NEG	NEG		LUPUS NEPHRITIS CLASS IV
37	IMMANUEL	35	NEG	NEG	NEG	MEDICAL RENAL DISEASE	DIABETIC NEPHROPATHY/RPS CLASS 2A/ACUTE INTERSTITIAL NEPHRITIS/SEVERE ARTERIOSCLEROSIS
38	NATARAJAN	31	NEG	NEG	NEG	MEDICAL RENAL DISEASE	IgA Nephropathy
39	NALINI	38	NEG	NEG	NEG		PROGRESSING GLOMERULONEPHRITIS/SCLEROSING GLOMERULONEPHRITIS
40	MAHAMED AYUB KHAN	32	NEG	NEG	NEG	MEDULLARY NEPHROCALCINOSIS	IgA Nephropathy
41	SAMUEL	30	NEG	NEG	NEG	CYSTITIS/MRD	DIABETIC NEPHROPATHY/RPS CLASS 2A/ACUTE INTERSTITIAL NEPHRITIS/SEVERE ARTERIOSCLEROSIS
42	LOGANATHAN	40	NEG	NEG	NEG	LT KIDNEY-MINIMAL CORTICAL CHANGE	MINIMAL CHANGE DISEASE
43	MESHACH	34	NEG	NEG	NEG		MEMBRANOUS NEPHROPATHY
44	SUBBULAKSHMI	21	NEG	NEG	NEG	B/L PLEURAL EFFUSION	ENDOCAPILLARY PROLIFERATIVE GLOMERYLONEPHRITIS ASS WITH INFECTION
45	MUTHUMARI	32	NEG	NEG	NEG		MEMBRANOUS NEPHROPATHY
46	MEENAKSHI	30	NEG	NEG	NEG	B/L MRD	IgA Nephropathy
47	RAMAKRISHNAN	21	NEG	NEG	NEG	B/L MRD	ENDOCAPILLARY PROLIFERATIVE GLOMERYLONEPHRITIS ASS WITH INFECTION
48	GOVIND	22	NEG	NEG	NEG		IgA Nephropathy
49	GANESAN	23	NEG	NEG	NEG		IgA Nephropathy
50	VANITHA	28	NEG	NEG	NEG		PROGRESSING GLOMERULONEPHRITIS/SCLEROSING GLOMERULONEPHRITIS